PUBLIC HEALTH REPORTS

VOL. 53 JANUARY 21, 1938

NO. 3

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

December 5, 1937-January 1, 1938

The accompanying table summarizes the prevalence of eight important communicable diseases based on weekly telegraphic reports from State health departments. The reports from each State are published in the Public Health Reports under the section "Prevalence of Disease." The table gives the number of cases of these diseases for the 4-week period ending January 1, 1938, the number reported for the corresponding period in 1936, and the median number for the years 1932–36.

DISEASES ABOVE MEDIAN PREVALENCE

Measles.—During the 4-week period ending January 1, 1938, 32,813 cases of measles were reported, as compared with 15,867 for the preceding 4 weeks. The number was almost five times that reported for the corresponding period in 1936 and more than three times the number in 1935. The current incidence was about 2.5 times the median incidence for 1932-36, which is a better comparison, since the number of reported cases of measles was unusually low in 1936 and also during the corresponding period in 1935. In the West South Central. Mountain, and Pacific regions the incidence was about normal, and in the New England region the number of cases was relatively low, but in all other regions the disease was unusually prevalent. In the Middle Atlantic and West North Central regions the number of cases reported was approximately four times the median incidence for this period in each region; in the East North Central region the incidence was about five times the median figure; and in the East South Central region the number of cases was about nine times the normal seasonal incidence for that region. The smallest increase was reported from the South Atlantic region, but even there the incidence was more than twice the average for the preceding years.

Scarlet fever.—The 18,928 cases of scarlet fever were only slightly in excess of the number reported for the corresponding period in 1936, which was a period of about average seasonal incidence. The larger number of the cases was reported from the Middle Atlantic and North Central States, with about a 20 percent increase over the

expected incidence in the New England States. In the South Atlantic, South Central, and Western groups the incidence was about normal for this season of the year.

Influenza.—The number of cases of influenza rose from approximately 4,500 for the preceding 4-week period to approximately 7,500 for the 4 weeks ending January 1, 1938. Such an increase in this disease is normally expected at this season of the year, and the current incidence compares very favorably with the experience of recent years, being slightly below that for the corresponding period in 1936 and only about 10 percent above the 1933–36 median. More than one-half of the total number of cases were reported from the South Central States.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period Dec. 5, 1937-Jan. 1, 1938, the number for the corresponding period in 1936, and the median number of cases for the corresponding period 1932-36 1

Geographic division	Current period	1936	5-year me- dian	Cur- rent peri- od	1936	5-year me- dian	Current peri-	1936	5-year me- dian	Cur- rent peri- od	1936	5-year me- dian
	D	iphthe	ria.	In	fluenz	B 3	M	[easles	•	Mer m	ingoco eningi	ecus tis
United States 1	2, 551	3, 031	4, 013	7, 481	7, 985	6, 760	32, 813	6, 801	13, 942	3,17	405	241
New England Middle Atlantic East North Central West North Central South Atlantic East South Central West South Central Mountain Pacific	52 329 477 229 574 269 401 80 140	40 355 459 184 841 367 500 65 220	113 531 855 395 788 409 655 95 184	97 494 316 1, 410 1, 415	26 152 849 308 2, 007 796 2, 940 441 466	133 748 353 2, 078 809 1, 453 228	13, 459 7, 593 4, 521	2, 413 1, 344 408 167 962 132 385 812 178	3, 589 1, 571 1, 075 1, 433 132 385	12 55 42 18 57 76 37 7	15 62 78 27 99 47 31 20 26	14 36 78 27 33 24 15 10
	Poli	lomyel	itis	Sca	rlet fe	ver	8:	mallpo	x	Тур	hoid fe	ver
United States 1	134	201	185	18, 928	17 , 63 0	18, 237	1, 338	636	518	497	754	754
New England Middle Atlantic East North Central West North Central South Atlantic East South Central West South Central Mountain Pacific	1 8 18 23 11 25 25 25 18	1 9 40 19 22 25 44 7	5 31 30 13 18 9 11 5	3, 157	1, 022 3, 385 5, 623 2, 955 1, 246 562 725 735 1, 377	1, 183 4, 241 6, 001 1, 801 1, 454 589 645 735 1, 221	0 0 417 438 6 36 32 249 160	0 76 61 226 5 1 10 111 146	0 1 93 171 11 15 46 76 145	24 89 29 37 92 27 121 32 46	18 103 85 68 154 127 94 59 46	30 103 124 62 160 93 149 59

¹ 48 States. Nevada is excluded, and the District of Columbia is counted as a State in these reports.
³ 44 States and New York City. The median is for the years 1933-36 only; the data for 1932 are not comparable.

Meningococcus meningitis.—For the 4 weeks ending January 1, 1938, the number of reported cases of meningococcus meningitis was 371, as compared with 405 and 436 for the corresponding period in the years 1936 and 1935, respectively. The current incidence is slightly above the median incidence for this period, as the years 1934, 1933, and 1932, which fall within the median period, were years of low seasonal incidence. The disease was above the seasonal expectancy in the

^{3 46} States. Mississippi and Georgia are not included.

Middle and South Atlantic Coast and South Central regions; below the median in the North Central and Pacific States and about normal in the New England and Mountain States.

Smallpox.—The number of cases of smallpox (1,338) reported for the current period was the highest recorded for the corresponding period in 6 years. The highest incidence was confined to States in the Mountain, Pacific, and North Central regions, with a slight rise above the normal seasonal incidence in the East South Central group of States. Of the total number of cases Indiana reported 267, Iowa 166, Illinois 121, Minnesota 115, and Idaho 101—more than one-half of the total cases occurring in those five States.

DISEASES BELOW MEDIAN PREVALENCE

Poliomyelitis.—The 134 cases of poliomyelitis reported for the current 4-week period represented a decrease from the total for the corresponding period in 1936 of about 35 percent, and was the lowest incidence reported for this period since 1932, when the incidence was approximately the same as for the current period. The West North Central region alone reported an increase over last year; in all other regions the incidence either closely approximated that of last year or fell considerably below it. The number of cases reported from the South Central States did not exceed that reported last year, but the incidence was considerably above the normal seasonal incidence for that area.

Diphtheria.—The incidence of diphtheria (2,551 cases) was the lowest recorded for this period in the 9 years for which these data are available. In the New England, North Central, and Mountain regions the current incidence was slightly above that for the corresponding period in 1936, but in relation to the 1932–36 median the current incidence was low in all sections of the country.

Typhoid fever.—Typhoid fever continued at a low level; 497 cases were reported for the current period, as compared with approximately 750 cases for the corresponding period in each of the years 1936 and 1935, and 1,039 in 1934. This disease has been considerably more prevalent in the West South Central region than it was in 1936, but that was apparently a "low" year for the disease in that region, and a better comparison is made with the 1932–36 median; the current incidence is about 20 percent below that. All other geographic divisions except the Pacific are low in relation to the 1932–36 median incidence.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ending January 1, 1938, based on data received from the Bureau of the Census, was 12.2 per 1,000 inhabitants (annual basis).

The average rate for the corresponding period in the 4 preceding years was also 12.2. In 1932 an epidemic of influenza raised the rate for this period to 13.3.

THE BLACKTONGUE-PREVENTIVE VALUE OF WHOLE WHEY, DELACTOSED WHEY, AND AMERICAN CHEESE *

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Goldberger, Wheeler, Lillie, and Rogers (1), showed that fresh skimmilk given to dogs on a blacktongue-producing diet in daily doses of approximately 30 cc per kilo of body weight prevented blacktongue in three out of five experimental animals for 1 year. Wheeler and Sebrell (2) conducted a similar experiment with canned evaporated milk in comparable daily doses (15 cc per kilo of body weight) and found a considerable delay in the onset of blacktongue in four out of five experimental animals. Booher and Hansmann (3), using a preparation obtained from a low lactose whey powder, were able to prevent blacktongue in one dog for 138 days, and successfully treated two dogs which had what were apparently mild symptoms of blacktongue.

Therefore, it was decided to test the blacktongue-preventive value of a whole whey powder, delactosed whey powder, and cheese.

			Nutrients					
	Article of diet	Quantity	Protein	Fat	Carbo- hydrate			
Casein (purified)	sinensis)3	 Grams 400 50 60 82	Grams 38. 6 10. 7 52. 0	Grams 18. 8 . 7	Grams 296. 0 30. 4			
Cottonseed oil		 32 30 15 10 3		30. 0 15. 0	82.0			
Total nutri		 	96. 3 40. 1	64. 5 26. 9	858. 4 149. 3			

TABLE 1.—Composition of basic blacktongue-producing diet No. 1231

¹ The corn meal, cowpeas (previously coarsely ground), and salt are stirred into water and cooked in a double boiler of enamelware for about 1½ hours. Then the other ingredients are well stirred in, the total weight being brought to 2,400 grams with water (so that 1 gram represents 1 calorie), and this finished mixture is served to the dog ad libitum.

² Whole maize meal (white) sifted as for human consumption.

³ The variety known as the California black-eyed pea.

⁴ Commercial casein leached for a week in daily changes of acidulated water, according to the method of McCollum, Simmonds, Shipley, and Park (Bull. Johns Hopkins Hosp., 33: 398 (1922)).

^{*}EDITORIAL NOTE.—This is another paper in a series of experimental studies designed to determine the blacktongue-preventive value of certain articles of food. The canine disease is thought to be identical with human pellagra.

EXPERIMENTAL

Through the courtesy of the Research Laboratories of Sealtest, Inc., and the Kraft Phoenix Cheese Corporation, we were furnished with a supply of whole whey powder, delactosed whey powder, and American cheese. The whole whey powder was obtained by the spray-drying process from milk produced in June. A portion of the same batch of whey was subjected to yeast fermentation in order to remove the lactose, and then spray-dried. The cheese was prepared in the same plant early in July. The analyses of the materials are given in table 2.

The yeast was not removed from the delactosed whey powder, and it was estimated to make up approximately 10 percent of the dry weight of the material. In the preparation of the whey powders it was calculated that 110 pounds of whole milk yielded 100 pounds of whey and 10 pounds of cheese, the whey having a dry weight of 7 pounds. After delactosing, the same quantity of whey yielded a preparation with a dry weight of 2.6 pounds. Hence, 1 gram of whole whey powder was recovered from 15.71 grams of whole milk; and 1 gram of delactosed whey powder was recovered from 42.31 grams of whole milk, while 1 gram of cheese was recovered from 11 grams of whole milk.

Riboflavin Acidity Ether Bour-Protein Lactose H₂O Ash as lactic extract quinacid Gamma Sherman per gram ³ units per gram Percent Percent Percent Percent Percent 72.74 .016 0.61 Whole whey. 12. 24 4.77 6.01 7.79 2.07 10.6 36.0 35. 82 30.09 4.77 1.38 1.79 32. 7 63. 5 Delactosed whey ... 31. 41 23.41 .000

TABLE 2.—Analyses of supplements:

¹ Determined by the light absorption method.

WHOLE WHEY POWDER

A group of five dogs (Nos. 261, 267, 292, 333, and 334) were placed on our basic blacktongue-producing diet No. 123, the composition of which is given in table 1. The whole whey powder was fed as a daily supplement mixed with a small portion of the ration. The original daily dose was 2.5 grams per kilo of body weight, which was calculated to represent the equivalent of 39.3 grams of whole milk.

¹ The analyses and data on the preparation of the whole whey and the delactosed whey powders were furnished us by Dr. Robert P. Myers and Dr. S. M. Weisberg of the Research Laboratories of Sealtest, Inc., Baltimore, Md. The cheese was analyzed in the Division of Chemistry of the National Institute of Health.

When symptoms of blacktongue developed in any animal, the supplement of all of the animals was increased until the daily dose of whole whey powder was 7.5 grams per kilo of body weight, or the equivalent of 117.8 grams of whole milk.

Three additional dogs (Nos. 320, 342, and 348) were added to the experiment later and received the 7.5 grams of whole whey powder per kilo of body weight throughout the experiment.

The significant details in regard to each of the experimental animals are as follows:

Dog No. 261

July 17, 1936: Begins diet 123 in good condition. Weight July 14 was 8.4 kilos. Begins daily supplement of approximately 2.5 grams of whole whey powder per kilo of body weight.

August 1: Fifteen days from the beginning of the experiment presents the first signs of an attack of blacktongue. An interrupted, red, band-like lesion on each side of the upper lip. The mucosa of the cheeks is diffusely injected, and the mucosa of the floor of the mouth is intensely red. The daily supplement of whole whey is increased to 5 grams per kilo of body weight.

August 3: The area of injection of the upper lip has become a continuous red band-like lesion. The mucosa of the cheeks and the floor of the mouth is very intensely injected and covered by thin pseudomembrane. The daily supplement of whole whey powder is increased to approximately 10 grams per kilo of body weight.

August 4: Weighs 7.5 kilos.

August 6: The buccal lesions have steadily progressed until the entire mucosa of the lips, cheeks, and the floor of the mouth is covered with thick pseudomembrane. There is considerable ropy salivation. The dorsal surface of the lateral margins of the tongue are red. The general condition is poor.

August 8: Moribund.

August 9: Found dead. Necropsy shows the typical lesions of advanced black-tongue.

Dog No. 267

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 6.25 kilos. Begins daily supplement of 16 grams of whole whey powder (2.5 grams per kilo).

August 1: The daily supplement of whole whey is increased to 31 grams (5 grams per kilo).

August 11: Shows faint reddening of the mucosa of the upper lip and cheeks of doubtful significance.

September 14: For the past forty days has shown transient, slight reddening of the buccal mucosa, suggestive of incipient blacktongue. Today the daily supplement of whole whey powder is increased to 46.5 grams (7.5 grams per kilo of body weight at beginning of experiment).

September 15: Weighs 6.6 kilos.

November 7-21: Faint red band-like lesion on each side of the upper lip and erythema of the mucosa of the cheeks and floor of mouth, characteristic of early blacktongue. The lesions spontaneously receded.

December 1-April 13, 1937: Transient, variable injection of the buccal mucosa.

April 21: Discontinues diet 123 and daily supplement of whole whey powder 278 days from the beginning of the experiment. During the test period there were repeated recurrent buccal lesions, suggestive of mild blacktongue, which never progressed to a definite acute attack.

Dog No. 292

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 8 kilos. Begins daily supplement of approximately 2.5 grams of whole whey powder per kilo of body weight.

August 1: Daily dose of whole whey increased to approximately 5 grams per kilo of body weight.

August 20: Thirty-four days from the beginning of the experiment presents first signs of an attack of blacktongue. Small reddened patches on the mucosa of each side of the upper lip opposite the canine teeth.

August 25: There is now a reddened band-like lesion about 1 inch wide on the left side of the upper lip, and several irregular patches of redness on the mucosa of the cheeks and the floor of the mouth.

September 1: The buccal lesions have receded.

September 8: Interrupted red band-like lesion on each side of the upper lip. Mild injection of the mucosa of the cheeks and floor of the mouth.

September 14: Whole whey powder supplement increased to approximately 7.5 grams per kilo of body weight.

September 15: Weighs 7.2 kilos.

September 16: The symptoms have progressed until now there is a continuous red band-like lesion extending along the mucosa on each side of the upper lip. The mucosa of the cheeks and the floor of the mouth is intensely red and covered by thick pseudomembrane. The scrotum presents a raised, sharply defined, desquamating area of dermatitis. There is a foul buccal odor and considerable salivation. Diarrhea.

September 17: The symptoms are progressing; the dog is in poor condition. Given 121.5 grams of whole whey powder dissolved in water by stomach tube on two occasions. Each dose was vomited.

September 18: Found dead. Necropsy reveals the typical lesions of advanced blacktongue.

Dog No. 333

July 17, 1936: Begins diet 123 in good condition. Weighed 6.8 kilos on July 14. Begins whole whey powder, 17 grams per day (2.5 grams per kilo).

July 28: Weighs 7.5 kilos.

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August 1: Whole whey powder supplement increased to 34 grams per day (5 grams per kilo of body weight at beginning of experiment).

August 20-September 17: Varying degrees of slight, transient redness of the buccal mucosa of doubtful significance.

September 14: The daily dose of whole whey powder is increased to 51 grams (7.5 grams per kilo of body weight at beginning of experiment).

November 12, 1936-January 24, 1937: Again had variable slight reddening of the buccal mucosa which receded. Passed blood by bowel today.

February 18: First signs of an acute attack of blacktongue 216 days from the beginning of the experiment. Injection of the mucosa of the cheeks and each side of the upper lip. Intense injection of the mucosa of the floor of the mouth.

March 9: Intensely red patches on the mucosa of each side of the upper lip. The mucosa of the cheeks and floor of the mouth is intensely injected.

March 12: There is now a bright red band-like area on the mucosa extending along each side of the upper lip. The mucosa of the cheeks is moderately injected, and the mucosa of the floor of the mouth and the lateral surface of the tongue is intensely injected.

March 13: The buccal lesions are more intensely injected. There is now pseudomembrane covering the mucosa of the cheeks. Removed from the experiment.

Dog No. 334

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 4.4 kilos. Begins daily supplement of approximately 2.5 grams of whole whey powder per kilo of body weight.

August 1: Whole whey powder supplement increased to approximately 5 grams per kilo of body weight.

August 25: Slight injection of the floor of the mouth. Has not eaten well for the last 30 days.

September 8: Weighs 3.1 kilos.

September 9: Found dead. Necropsy: Extensive bilateral bronchopneumonia.

Dog No. 320

September 20, 1936: Begins diet 123 in good condition. Weight on September 15 was 5.0 kilos. Begins daily supplement of 37.5 grams whole whey powder (7.5 grams per kilo).

November 7: Has been observed at coprophagy.

November 17-December 1: Varying degrees of redness of the mucosa of the upper lip, cheeks, and floor of the mouth which did not become severe enough to warrant a definite diagnosis of acute blacktongue.

January 5, 1937: First sign of an attack of acute blacktongue 107 days from the beginning of the experiment. Faint injection of the mucosa of the upper lip and cheeks. Intense reddening of the mucosa of the floor of the mouth.

January 12: The mucosa of the right side of the upper lip is intensely injected, while that of the left side has a bright red patch opposite the canine tooth. The mucosa of the cheeks, floor of the mouth, and lateral surface of the tongue is intensely injected. There is some pseudomembrane on the mucosa of the right side of the upper lip. Weighs 4.5 kilos.

January 13: The buccal lesions have progressed and there is now pseudomembrane on the mucosa of the upper lip and both cheeks. Removed from the experiment.

Dog No. 348

September 20, 1936: Begins diet 123 in good condition. Weight on September 15 was 6.5 kilos. Begins daily supplement of approximately 7.5 grams of whole whey powder per kilo of body weight.

November 3: First sign of an acute attack of blacktongue 44 days from the beginning of the experiment. A diffuse injection of the mucosa of the cheeks and floor of the mouth. Weighs 6.25 kilos.

November 21: There are brilliant red patches on the mucosa of each side of the upper lip opposite the canine tooth. The remainder of the mucosa of the upper lip is diffusely injected. The mucosa of the cheeks, the floor of the mouth, and the lateral surfaces of the tongue is moderately injected.

November 25: The buccal lesions have steadily progressed and pseudomembrane now covers the reddened mucosa of the cheeks and the upper lip. Removed from the experiment.

Dog No. 348

September 20, 1936: Begins diet 123 in good condition. Weight on September 15 was 5.2 kilos. Begins daily supplement of 39 grams whole whey powder (7.5 grams per kilo).

April 21, 1937. During the 213-day test period the animal has had recurrent transient injection of the buccal mucosa of doubtful significance. Today the mouth is entirely clear. Discontinues diet 123 supplemented by whole whey powder.

Summary.—Three of the five dogs (Nos. 261, 292, and 233) starting the experiment on the 2.5 grams per kilo of body weight supplement of whole whey powder developed blacktongue in 15 days, 34 days, and 216 days, respectively, from the beginning of the experiment. One animal (dog No. 234) died of pneumonia, and one (dog No. 267) showed fleeting redness of the buccal mucosa, suggestive of incipient blacktongue, which first appeared 25 days from the beginning of the experiment. After the whey powder was increased to 7.5 grams per kilo of body weight, he finally completed a total test period of 278 days, with only recurrent fleeting symptoms suggestive of incipient blacktongue.

Two of the three dogs (Nos. 320 and 342) that began the experiment on a daily supplement of 7.5 grams of whole whey powder per kilo of body weight developed blacktongue in 107 days and 44 days, respectively, from the beginning of the experiment. The remaining animal (dog No. 348) completed a test period of 213 days, during which time he showed recurrent, fleeting redness of the buccal mucosa, suggestive of early blacktongue.

Therefore, whole whey powder in the quantity given must be regarded as a poor source of the blacktongue-preventive factor. Since it is impracticable to give doses larger than 7.5 grams per kilo of body weight, it appears that whole whey powder does not contain enough of the blacktongue-preventive factor to be of any practical significance.

DELACTOSED WHEY POWDER

A group of five dogs (Nos. 266, 288, 330, 332 and 335) were placed on our basal blacktongue-producing diet No. 123, the composition of which is given in table 1. The delactosed whey powder was fed as a daily supplement mixed with a small portion of the ration. The original daily dose was 1 gram per kilo of body weight, which was calculated to represent the equivalent of 42.31 grams of whole milk.

When symptoms of blacktongue developed in any animal, the supplement of all of the animals was increased until the daily dose of delactosed whey powder was 3 grams per kilo of body weight, or the equivalent of 126.9 grams of whole milk.

The significant details in regard to each of the experimental animals are as follows:

Dog No. 266

July 17, 1956: Begins diet 123 in good condition. Weight on July 14 was 7 kilos. Begins daily supplement of approximately 1 gram of delactosed whey powder per kilo of body weight.

August 11: Delactosed whey powder supplement increased to approximately 2 grams per kilo of body weight.

September 27: Since August 11 has shown varying degrees of redness of the buccal mucosa, suggestive of incipient blacktongue, which spontaneously receded. Today delactosed whey powder supplement increased to approximately 3 grams per kilo of body weight.

October 3: First sign of an acute attack of blacktongue 78 days from the beginning of the experiment. A faint, red band-like lesion on the mucosa of each side of the upper lip. Mucosa of the floor of the mouth diffusely injected.

October 6: There is a red band-like lesion about one-half inch wide on the mucosa of each side of the upper lip. Small necrotic areas are scattered along this injected lesion. The mucous membrane of the cheeks and the floor of the mouth are intensely injected, and there is much pseudomembrane on the cheeks. Weighs 7 kilos.

October 7: Lesions have progressed to pseudomembrane formation on the mucosa of the upper lip and the floor of the mouth, as well as on the cheeks. There are numerous areas on the buccal mucosa showing punctate hemorrhages. General condition is poor.

October 8: The animal's condition is progressively worse. Has extensive necrosis of the mucous membrane of the lips and cheeks. The margins of the tongue are quite red, and the mucosa of the floor of the mouth shows considerable necrosis and pseudomembrane. There is much salivation.

October 10: Found dead. Necropsy shows the typical lesions of advanced blacktongue.

Dog No. 288

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 7.8 kilos. Begins daily supplement of approximately 1 gram of delactosed whey powder per kilo of body weight.

August 8-17: Varying degrees of slight reddening of the buccal mucosa which spontaneously receded.

August 11: Delactosed whey powder supplement increased to approximately 2 grams per kilo of body weight.

September 26: First signs of an acute attack of blacktongue 71 days from the beginning of the experiment. Large, bright red patches on each side of the upper lip opposite the canine teeth. The mucosa of the cheeks and floor of the mouth is fiery red. Given 4 grams per kilo of body weight of delactosed whey today only.

September 27: Delactosed whey powder supplement increased to approximately 3 grams per kilo of body weight.

September 29: Weighs 7.8 kilos.

September 30: Continues to have bright red areas on each side of the upper lip. The cheeks and the floor of the mouth continue very red, and there is a reddened patch on the dorsum of the tip of the tongue.

October 1: Lesions have progressed and pseudomembrane now covers the reddened areas on the mucosa of the upper lip and cheeks. The mucosa of the floor of the mouth remains intensely injected.

October 3: The buccal lesions are progressing, and there is considerable salivation.

October 5: General condition is poor. There is much salivation and extensive pseudomembrane on the mucosa of the upper lip and cheeks. The lateral margins of the tongue are bright red.

October 6: Moribund. Found dead later in the day. Necropsy shows the typical lesions of advanced blacktongue.

Dog No. 330

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 7 kilos. Begins daily supplement of 7 grams delactosed whey powder (1 gram per kilo).

August 11: Delactosed whey powder supplement increased to 14 grams (2 grams per kilo).

September 27: Delactosed whey powder supplement increased to 21 grams (3 grams per kilo).

November 17, 1936-January 12, 1937: Showed transient, slight injection of the mucosa of the upper lip, or cheeks, or both.

April 21: Discontinues diet 123 and whole whey powder supplement 278 days from the beginning of the experiment.

Dog No. 332

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 9 kilos. Begins daily supplement of approximately 1 gram of delactosed whey powder per kilo of body weight.

August 11: Weighs 10 kilos. Daily dose of delactosed whey powder supplement increased to 2 grams per kilo of body weight at beginning of experiment.

September 27: Delactosed whey powder supplement increased to 3 grams per kilo of body weight at beginning of experiment.

October 13: Weighs 10.4 kilos.

October 17: 92 days from the beginning of the experiment shows first signs of an acute attack of blacktongue. The mucosa of the cheeks and the floor of the mouth is quite red.

October 19: Buccal lesions have progressed rapidly. There are now large, brilliant red patches covered with thick pseudomembrane on the mucosa of each side of the upper lip opposite the canine teeth. There is also an interrupted red band on the mucosa of the upper lip on each side. The mucosa of the cheeks and floor of the mouth is very red, and covered by pseudomembrane. Removed from the experiment.

Dog. No. 335

July 17, 1936: Begins diet 123 in good condition. Weight on July 14 was 8.5 kilos. Begins daily supplement of 8.5 grams delactosed whey powder (1 gram per kilo).

August 11: Delactosed whey powder supplement increased to 17 grams daily (2 grams per kilo).

September 26: Given 4 grams per kilo of delactosed whey powder supplement today only.

September 27: Delactosed whey powder supplement increased to 25.5 grams daily (3 grams per kilo).

January 30, 1937: Since December 22 has shown recurrent, transient injection of the mucosa of the upper lip, cheeks, and floor of the mouth of doubtful significance, which spontaneously receded. Passed small amount of blood by bowel today.

February 2: Weighs 9.3 kilos.

February 9: First signs of an acute attack of blacktongue 207 days from the beginning of the experiment. A red patch on the mucosa on each side of the upper lip and on each cheek. The mucosa of the floor of the mouth is slightly injected. Weighs 8 kilos.

February 10: The reddened patch on the mucosa of the left side of the upper lip is larger, and more intensely injected; that on the mucosa of the right side of the upper lip has become a brilliant red band extending the length of that side of the lip. The mucosa of the cheeks is intensely reddened and covered with pseudomembrane. The mucosa of the floor of the mouth is intensely injected, and the soft palate is quite red. Removed from the experiment.

Summary.—Four of the five dogs (Nos. 266, 288, 332 and 335) developed the first signs of blacktongue in 78 days, 71 days, 92 days, and 207 days, respectively, from the beginning of the experiment. The remaining dog (No. 330) showed transient buccal symptoms suggestive of incipient blacktongue 123 days from the beginning of the experiment, which receded and reappeared throughout the experiment, although the animal completed an experimental period of 278 days without developing a definite acute attack of blacktongue.

Therefore, delactosed whey powder in the quantity given must be regarded as a poor source of the blacktongue-preventive factor. It appeared to be inadvisable to increase the daily amount of the delactosed whey to more than 3 grams per kilo of body weight, since the interpretation of the results might be difficult because of its 10 percent yeast content.

AMERICAN CHEESE

A group of five dogs (Nos. 352, 358, 363, 365, and 367) were placed on our basal blacktongue-producing diet No. 123. The cheese was broken up in small pieces and served separately before the ration was given. It was readily taken without difficulty. The original daily dose was 5 grams per kilo of body weight, which was calculated to represent the equivalent of 55 grams of whole milk.

On the first appearance of the symptoms of blacktongue in the experimental animals in 22 days (dog No. 363) the daily supplement of cheese was increased to 10 grams per kilo of body weight. The significant details in regard to each of the experimental animals are as follows:

Dog No. 352

January 6, 1937: Begins diet 123 in good condition. Weight yesterday was 7.5 kilos. Begins daily supplement of 5 grams of American cheese per kilo of body weight.

January 29: Weighs 8.7 kilos. Daily cheese supplement increased to 80 grams (approximately 10 grams per kilo of body weight)...

February 11: Left 30 grams of cheese supplement.

February 16: First signs of blacktongue 41 days from the beginning of the experiment. The floor of the mouth is diffusely injected and there is a faint red patch on the left side of the upper lip. Weighs 9.5 kilos.

March 6: Left 40 grams of cheese supplement.

March 9: Buccal lesions have slowly but steadily progressed and there is now a red band-like lesion on the mucosa of each side of the upper lip. The mucosa of the cheeks and floor of the mouth is moderately injected.

March 11: Left 50 grams of cheese supplement.

March 13: Left 65 grams of cheese supplement.

March 14: Left all of cheese supplement.

March 15: Buccal lesions have continued to increase in severity. There are many small hemorrhagic and ulcerated areas on the mucosa of the upper lip which is intensely red. The mucosa of the cheeks is intensely injected and covered with pseudomembrane. The mucosa of the floor of the mouth is intensely injected. There is considerable salivation, and the saliva is blood tinged. Had bloody stool today. Removed from the experiment.

Dog No. 358

January 12, 1937: Begins diet 123 in good condition. Weighs 7.2 kilos. Begins daily supplement of 36 grams of American cheese (5 grams per kilo).

January 29: Daily cheese supplement increased to 72 grams (10 grams per kilo).

February 7: Passed blood by bowel.

February 11: Passed blood-stained material by bowel.

February 10-April 2: Left an average of 16 grams of cheese supplement daily.

April 6: First signs of an acute attack of blacktongue 84 days from the begin-

April 6: First signs of an acute attack of blacktongue 84 days from the beginning of the experiment; a slight redness of the mucosa of the upper lip. Weighs 9.4 kilos.

April 9: Left 34 grams of cheese.

April 10: The buccal lesions have progressed very rapidly and the mucosa of the upper lip is intensely reddened and covered with pseudomembrane. The mucosa of the cheeks is covered with dense pseudomembrane, and the mucosa of the floor of the mouth is intensely injected. There is a foul buccal odor, and much salivation. Removed from the experiment.

Dog No. 363

January 6, 1937: Begins diet 123 in good condition. Weight yesterday was 8.9 kilos. Begins daily supplement of approximately 5 grams of American cheese per kilo of body weight.

January 26: Weighs 10 kilos.

January 28: First signs of an acute attack of blacktongue 22 days from the beginning of the experiment: A sharply outlined red bandlike lesion on the mucosa of each side of the upper lip. The mucosa of the cheeks is moderately injected, and the mucosa of the floor of the mouth is rather intensely injected.

January 29: The red band on the mucosa of each side of the upper lip is wider. The cheeks are covered with thin pseudomembrane, and the mucosa of the floor of the mouth is intensely red. There is a foul buccal odor. Removed from the experiment.

Dog No. 365

January 6, 1937: Begins diet 123 in good condition. Weight yesterday was 6.8 kilos. Begins daily supplement of approximately 5 grams of American cheese per kilo of body weight.

January 29: Weight 7.2 kilos. American cheese supplement increased to approximately 10 grams per kilo of body weight.

February 16-March 25: Has had transient and varying degrees of injection of the mucosa of the cheeks and the floor of the mouth, which spontaneously receded.

March 16 to July 10: Left on an average of 4.8 grams of cheese supplement per day.

July 27: Weighs 8.4 kilos.

July 30: Discontinued diet 123 and cheese supplement in good condition 205 days from beginning of experiment. At no time did the animal have symptoms that could be definitely diagnosed as blacktongue.

Dog No. 367

January 6, 1937: Begins diet 123 in good condition. Weight yesterday was 8.4 kilos. Begins daily supplement of approximately 5 grams of American cheese per kilo of body weight.

January 29: Daily supplement of American cheese increased to approximately 10 grams per kilo of body weight (90 grams). Weighs 9 kilos.

March 1-May 18: Left on an average of 24.9 grams of cheese per day.

May 18: First sign of an acute attack of blacktongue 132 days from the beginning of the experiment: Four discrete red patches on the mucosa of the upper lip. The mucosa of the cheeks is intensely injected, and small areas are covered with pseudomembrane. The mucosa of the floor of the mouth is intensely injected.

May 21: The buccal lesions have faded spontaneously and the mouth is now practically normal.

May 18-24: Left an average of 27 grams of cheese supplement per day.

May 24: There are several bright red patches on the mucosa of the upper lip. The mucosa of the cheeks and the floor of the mouth shows a rather intense, diffuse injection.

May 26: Has a continuous bright red bandlike lesion on the mucosa of the upper lip. The mucosa of the cheeks is intensely injected, and areas are covered with pseudomembrane. The mucosa of the floor of the mouth is intensely injected. Had a semiliquid tarry stool today. Removed from the experiment. Weighs 9.2 kilos.

Summary.—Four of the five dogs (Nos. 352, 358, 363 and 367) showed the first signs of an acute attack of blacktongue in 41 days, 84 days, 22 days, and 132 days, respectively, from the beginning of the experiment. The remaining animal (No. 365) had recurrent, transient attacks of reddening of the buccal mucosa, suggestive of incipient blacktongue, beginning 41 days from the beginning of the experiment, but completed the experimental period of 205 days without having a definite acute attack of blacktongue.

Therefore, American cheese in the quantity given is a poor source of the blacktongue-preventive factor. The quantity used (10 grams per kilo) apparently approached the limit which the animals would tolerate, and it did not appear that findings with a larger amount than this would be of any practical significance.

CONCLUSION - 3

Whole whey powder, delactosed whey powder, and American cheese are poor sources of the blacktongue- (pellagra-) preventive factor.

REFERENCES

(1) Goldberger, Joseph, Wheeler, G. A., Lillie, R. D., and Rogers, L. M.: A study of the blacktongue-preventive action of 16 foodstuffs, with special reference to the identity of blacktongue of dogs and pellagra of man. Pub. Health Rep., 43: 1385 (1928).

(2) Wheeler, G. A., and Sebrell, W. H.: The blacktongue (canine pellagra) preventive value of fifteen foodstuffs. Natl. Inst. of Health Bull. No. 162, Part I., page 1, Govt. Printing Office, Washington, D. C., 1933.
(3) Booher, L. E., and Hansmann, G. H.: Vitamin G concentrates as preventives against blacktongue, with parallel studies of some concentrates in diets of white rats. Am. J. Physiol. 114: 429 (1936).

RIBOFLAVIN DEFICIENCY IN DOGS

By W. H. SEBRELL, Surgeon, and R. H. ONSTOTT, Passed Assistant Surgeon, United States Public Health Service, National Institute of Health

Sebrell, Onstott, and Hunt (1) have presented preliminary evidence that riboflavin 1 deficiency produces a symptom complex in dogs, characterized by sudden collapse and coma, promptly followed by death, unless treated early with adequate doses of riboflavin. The striking similarity of symptoms and the finding of yellowish coloration of the liver at autopsy in one animal suggested that the condition previously described from this laboratory by Sebrell (2, 3) as "yellow liver" was due to a riboflavin deficiency.

The experimental diets Nos. 330 and 341 used by Sebrell (3) contained relatively large amounts of rice polishings. Diet 330 contained 180 grams of rice polishings per 2,400-calorie ration, while diet No. 341 contained 400 grams per 2,400-calorie ration. All of the five dogs on diet No. 330 died, and at autopsy all presented the characteristic findings of "yellow liver." The time of onset of symptoms in these dogs was 162, 175, 240, 275, and 335 days, respectively, from the beginning of the experiment—an average of 238 days.

On the other hand, three dogs placed on diet No. 341 completed 1 year on the diet in good condition, were killed, and at autopsy failed to show any signs of "yellow liver."

The time of appearance of the symptoms of riboflavin deficiency in the four dogs on diet 123, supplemented with rice bran filtrate K-37-A, reported by Sebrell, Onstott, and Hunt (1), was 171, 196, 137, and 172

¹ Upon the recommendation of its Committee on No menclature, the Council on Pharmacy and Chemis' try of the American Medical Association has adopted the name "Riboflavin" for the compound previously known as vitamin B₂. The name indicates that this compound is a ribose derivative of isoalloxazine, and prevents confusion with many synthetic products also called flavins but which are devoid of ribose.

The Council states that, while flavin was first isolated from egg white, the evidence is rather convincing that the flavins obtained from egg white, milk, liver, pancreas, and a number of other sources are identical in structure and manifest the properties of vitamin B₂. There appears to be a fairly general agreement among investigators in the field of vitamins who have discussed this question of nomenclature that the name "flavin" should be given to a water-soluble pigment that has been demonstrated to be necessary for the normal nutrition of the rat and for growing chicks.

The report of the Council appeared in the Journal of the American Medical Association, vol. 108, No.16, p. 1340, April 17, 1937.

days, respectively—an average of 169 days from the beginning of the experiment.

With these facts in mind, it was decided to conduct an experiment similar to some of the earlier work on "vellow liver," treating the animals with a preparation of riboflavin when symptoms of "yellow liver" appeared.

EXPERIMENTAL

Diet No. 465 was constructed to contain 135 grams of rice polishings per 2,400-calorie ration. If, as according to Munsell (4) and interpreted by Cowgill (5), rice polishings contain 4.2 international units of B₁ per gram, each ration of diet No. 465 contains 527 international units of B₁ or 175.67 international units for an average 800-gram serving. Calculated according to Cowgill (5),2 the requirement of a 5-kilo dog is 7.49 international units per day, that of a 10-kilo dog is 17.64 international units per day, and that of a 15-kilo dog is 34.67 international units per day. Thus, it would appear that our diet 465 contains an adequate amount of vitamin B, for the dogs used in this experiment.

TABLE 1.—Composition of diet No. 465 1

	Percent			Nutrients				
Article of diet	of dry weight	Quantity	Protein	Fat	Carbo- hydrate			
Rice polishings * Casein (purified) * Casein (purified) * Cornstarch (commercial) Cottonseed oil Cod liver oil Salt mixture *	23.5 13.9 3.0 41.7 11.5 2.6 3.8	Grams 135. 0 80. 0 17. 0 240. 0 66. 0 15. 0 22. 0	Grams 19. 2 72. 5	Grams 0. 3 66. 0 15. 0	Grams 91. 7 17. 0 216. 0			
Total Calories	100.0	575. 0 2, 400. 0	91. 7 368. 0	87. 3 732. 0	824. 7 1, 300. 0			

¹ The rice polishings, cornstarch, and cottonseed oil are stirred into tap water and cooked in a double boiler of enamelware for about 1½ hours. The other ingredients are well stirred in, the total weight being brought to 2,400 grams with water (so that 1 gram represents 1 calorie), and this finished mixture is served to the dogs ad libitum.

Five dogs (Nos. 347, 357, 360, 362, and 364) were placed on diet No. 465, the composition of which is given in table 1. On the appearance of symptoms similar to those previously observed in this labora-

to the dogs ad nortum.

² Commercial rice polishings extracted with ether until the percolate is colorless.

³ Commercial casein leached for a week in daily changes of acidulated water, according to the method of McCollum, Simusonds, Shipley, and Park (Bull. Johns Hopkins Hosp., 33: 398 (1922).)

⁴ Prepared according to the method of Osborne and Mendel (J. Biol. Chem., 37: 557 (1919)).

³ Vitamin B₁ (international units per day) = $\frac{0.000076 \text{ (weight gra 4/3)}}{2}$.

tory in connection with "vellow liver" of dogs (collapse or coma). each dog was treated with a preparation of riboflavin, either by intramuscular or intraperitoneal injection. Several of the dogs became noticeably anemic during the experiment; and beginning on the 111th day after the experiment was started, weekly determinations of red blood cells and hemoglobin were made on all of the experimental These results are shown in table 2. animals.

TABLE 2.—Red blood cells and hemoglobin ...

	Dog N	o. 347	Dog N	To. 3 57	Dog N	o. 360	Dog N	o. 262	Dog N	o. 364
Day of experiment	Red blood cells	Hemo- globin ¹	Red blood cells	Hemo- globin ¹	Red blood cells	Hemo- globin ¹	Red blood cells	Hemo- globin ¹	Red blood cells	Hemo- globin ¹
118	6, 900, 000 6, 800, 000 97,800,000	13. 5 14. 0 13. 8 13. 6	2, 600, 000	13. 0	\$ 6, 500, 000 5, 800, 000 4, 500, 000 5, 300, 000 5, 700, 000 6, 000, 000 5, 600, 000 6, 200, 000 6, 200, 000 5, 700, 000	11. 5 11. 4 9. 5 10. 5 9. 6 11. 5 12. 8 10. 6 10. 3 10. 8 11. 0	5, 900, 000 5, 200, 000 6, 600, 000 6, 800, 000 6, 400, 000 7, 000, 000 5, 600, 000 6, 100, 000 6, 700, 000 6, 500, 000 6, 500, 000 6, 500, 000 6, 500, 000 6, 500, 000 6, 500, 000	12. 5 14. 0 10. 6 13. 3 12. 6 11. 8 15. 2 15. 0 13. 4 14. 8 14. 0 13. 6	4, 400, 000 45,000,000 3, 590, 000 5, 100, 000 5, 400, 000 4, 600, 000 4, 900, 000 2, 900, 000 3, 300, 000 73,600,000 3, 600,000 3, 700, 000	8. 4 6. 4 7. 8 5. 8 5. 1 3. 8 4. 4 4. 9
211 216 217 217					5, 100, 000 4, 800, 000	10. 2	6, 400, 000 7, 300, 000	l	3, 7 90 , 000 4, 200 , 000	4. 7 6. 0
224 230 231 237					5, 000, 000 5, 000, 000 5, 600, 000	8. 5 9. 0	6, 600, 000 6, 000, 000		4, 90 0, 000 5, 400, 000	5. l
245 147 1852					5, 100, 000 10 4,100,000	8. 2	6, 700, 000 6, 800, 000		4, 600, 000 5, 700, 000	7. 5

¹ Hemoglobin determinations according to Newcomer (J. Biol. Chem., 37: 465 (1918); 55: 569 (1923)).

When it was noticed that bradycardia and a sinus type of arrhythmia was a common finding, routine heart rate determinations were These results are shown in table 3.

Acute attack 121st day. Died 122d day.

Acute attack 118th day. 4 Acute attack 125th day.

Acute attack 140th day; died.

[•] Acute attack 169th day.

Acute attack 188th day. Acute attack 203d day.

[·] Acute attack 235th day.

¹⁶ Acute attack 255th day.

Furnished through the courtesy of Mr. John Hart of the Winthrop Chemical Co., Inc.; packaged in glass sealed ampules containing 2 cc of a 0.05 percent solution and designated as L. F. 356.

TABLE 3 .- Heart rates

Day of experiment	Dog No. 347	Dog No. 857	Dog No. 860	Dog No. 362	Dog No. 364
18 21		M. 40. 1 60. 818	1 80		
22 25		160			11
26					14
32	114			140	12
39	104		1 88	112	14
4048			88	120	12
1 053			188	104	16
60			104	184	i
67			1 .00	96	12
8 0 70			170, 1888		
			1 86	120	18
75 81			1 96	104	14
88					158, 82, 98,
89					14
95				120	13
02 03			1 86, 1 76	110	-14
03			1 88 (211 days)	120	14
16			88 (217 days)	92	18
23			84 (224 days)	108	12
<u>3</u> 0			88 (231 days)	88	
35			1 80, 174, 164, 184 172 (238 days)	120	
37			96 (247 days)	108	13 12
52			96	116	12
55			164, 184, 180, 1110		

Rates in italics were taken during an acute attack.

The significant details in regard to each of the experimental animals are as follows:

Dog No. 347

December 8, 1936: Begins diet No. 465 in good condition. Weighs 8.4 kilos.

January 26, 1937: The skin of the scrotum, flanks, and both axillae is reddened.

January 30: The redness of the skin lesions is fading. There is desquamation of the skin of the flanks, axillae, and scrotum.

February 4: Red desquamating lesions of the abdomen and the scrotum. The lesion on the scrotum now appears very similar to the scrotal lesion described in blacktongue.

February 9: The skin lesions have cleared up.

April 27: 140 days from the beginning of the experiment.

9:30 a. m.: Appears entirely normal except for a dry, scaly dermatitis of the scrotum. Weighs 8.7 kilos.

11:35 a. m.: Found lying on side in a semicomatose condition. Eyes open, able to lift head, reflexes hyperactive, slightly spastic, heart rate 98, respirations 10 per minute. Has exaggerated sinus type cardiac arrhythmia. Given 1 mg riboflavin (L. F. 356), diluted to 10 cc with normal saline, intramuscularly.

12:30 p. m.: Is in a deep coma. There is no response to mechanical stimuli.

2:45 p. m.: Is in deep coma. Respirations shallow and rapid. The heart rate is 58 per minute, with slight irregularity. Given 1 mg riboflavin (L. F. 356), diluted to 10 cc with normal saline, intramuscularly.

¹ Marked arrhythmia of the sinus type.

Following administration of atropin sulphate.
 Immediately after recovery from acute attack.

4:15 p. m.: Continues in a deep coma. Heart rate is 72 per minute, continues slightly irregular. Respirations are slow, have become shallow, jerky, and labored. No response to mechanical stimuli.

5 p. m.; Condition has become steadily worse. Coma is very deep. Respirations are quite shallow.

10 p. m.: Found dead. Placed in cold room.

April 28: Necropsy at 9:15 a. m.

NECROPSY

Gross findings: The dog is well nourished. The nose and mouth are stained with fluid which appears to be bloody. The skin of the abdomen is slightly erythematous, and there is a dry, branny, scaly dermatitis of the scrotum. Brain and cord appear normal, although the meninges are covered with a fibrinous blood-tinged exudate. The lower lobe of the right lung is markedly congested. There are congested areas throughout the upper lobe. The left lung has several small areas of congestion. Liver is of normal size and consistency, and presents a yellowish, mottled surface. The cut section is uniform and yellowish brown in color.

Intestines: There is an intussusception of the proximal jejunum and duodenum into the pyloric end of the stomach. The mucosa of the duodenum and pylorus is quite red, and there is some blood stained mucus in the stomach.

(Microscopic pathology under dog No. 357.)

Discussion.—The failure of this dog to respond to treatment with riboflavin (L. F. 356) may have been due to a combination of two factors. First, the initial dose of riboflavin was only 1 mg, and there was an interval of 3 hours and 10 minutes before a second dose of 1 mg was given. At the time the second dose was given the dog appeared moribund, so that the effective amount of riboflavin administered may have been too small. Second, the riboflavin was diluted to 10 cc with normal saline. This dilution may have rendered the absorption materially slower, so that in effect the dog may have received little flavin.

Dog No. 357

December 8, 1936: Begins diet 465 in good condition. Weighs 4.4 kilos. April 7, 1937: Apparently normal in late afternoon.

April 8: 121 days from the beginning of the experiment, found apparently dead at 8:30 a. m. The animal was cold, and there were no visible respiratory movements. At 8:40 a. m. the animal was reexamined, and was found to be alive. There were no visible respiratory movements, but the heart was beating slowly. The animal was immediately given 2 mg of riboflavin (L. F. 356) intraperitoneally.

9 a. m.: Dog breathing, respirations 10 per minute, shallow, but regular. Heart rate 41 per minute, regular. Given 2 mg of riboflavin (L. F. 356) intramuscularly.

10 a. m.: In deep coma, heart rate remains about 40 per minute. Electrocardiogram 4 is normal except for inversion of the T-wave, and the slow heart rate.

Obtained through the courtesy of Capt. H. W. Smith of the Naval Medical School, Washington, D. C.

11 a. m.: Animal remains in deep coma. There is no movement, except respiration. Heart rate is 60 per mniute, but is quite irregular. Respiration 12 per minute.

1:80 p. m.: Observed having a convulsive seizure consisting of generalized tremors, clonic spasms, and opisthotonus.

5 p. m.: Still semicomatose, but attempts to rise and move about. Appears unable to move hindquarters, pulling self about the room with forelegs.

11 p. m.: Animal is in deep coma. Does not respond to stimuli.

9:30 a. m.: Dog still alive, but is in deep coma. Heart rate 160 per minute, regular. Respiration slow and shallow. Given 1 mg of riboflavin (L. F. 356) in 200 cc of normal saline, intraperitoneally.

2. Dog dead; without regaining consciousness. During the entire period of coma passed no urine.

MECROPSY

Gross findings: All tissues examined appear entirely normal with the following exceptions: Brain—Mild hyperemia of the meninges of the vault. Lungs— Moderate hypostatic congestion. Liver—The liver is normal in size and consis-A marked yellowish mottling is present. No areas appear normal, some areas are almost a pure yellow color, others mottled yellow and pink. section reveals an almost uniform vellowish gray color. Bladder—Collapsed and contracted. Contains no urine.

HISTOLOGIC PATHOLOGY

(By Surgeon R. D. Lillie)

In two dogs studied histologically the brain and cord showed extensive and very moderate nerve cell degeneration, respectively, with tigrolysis, nerve cell vacuolation, and deposition of fine fat droplets in the cytoplasm. In the one (347) with the more extensive cellular degeneration Marchi preparations showed also some swelling, distortion, and blackening of a more or less numerous minority of myelin sheaths in certain levels of the pyramidal tracts in brain and cord, in parts of the median longitudinal bundles and brachium pontis in the pons, in radicular or root fibers of the glossopharyngeus and accessorius nerves, and in part of the fasciculus cuneatus in the cord. Exudative inflammatory changes were absent.

The livers showed diffuse deposition of fine and medium droplets of neutral fat in the liver cells as well as the normal fat in the bile duct epithelium. In one dog (347) there were also scattered coagulated necrotic liver cells, more in the lobule centers.

This dog showed also some tubular and glomerular necrosis while the other presented only a moderate vacuologranular degeneration of the convoluted tubules and the normal fatty epithelium in the coarse loop tubules.

Nodular hemorrhagic consolidation was present in the lungs of both, more extensive in the one (347) than in the other. muscle was normal in both. The spleen was anemic and atrophic.

Femoral marrow was largely fatty in both, vertebral and sternal as well in the one (357) in which it was studied. Hypophyses showed chromophil preponderance in the pars anterior. In 357, stomach, large and small intestine and bladder showed no significant lesions.

The changes noted here are similar to those described previously by Lillie and Sebrell (6) in "yellow liver" of dogs. They are apparently more acute and accordingly more purely degenerative in so far as the central nervous system is concerned, but otherwise suggest the neuropathologic picture described by Zimmerman and coworkers (7, 8).

Discussion (dog No. 357).—This dog was found comatose early in the morning, and was apparently dead when removed from the kennel. It was evident that he had been comatose for some time, as his body was quite cold. The failure of this dog to respond to treatment may be explained on the assumption that the condition had progressed too far when treatment was started. The dog rallied for a short period, and lived for 30% hours after treatment was started. This relatively long survival period suggests that the treatment was not entirely without effect. The findings in the electrocardiogram obtained on this dog indicate that the bradycardia and the exaggerated sinus arrhythmia observed are due to some extracardiac cause.

Dog No. 360

December 8, 1936: Begins diet 465 in good condition. Weighs 6.9 kilos.

March 16, 1937: Weighs 7.3 kilos. Appears entirely normal with the exception of noticeable pallor of the buccal mucous membranes.

April 5: 118 days from the beginning of the experiment.

10 a. m.: Appears entirely normal with the exception of some pallor of the buccal mucosa.

3:20 p. m.: Seen standing in kennel.

3:30 p. m.: Found in deep coma. Heart rate 80 per minute, and there is an exaggerated sinus type of cardiac arrhythmia. Respiration 12 per minute, regular. Given 5 mg of riboflavin (L. F. 356) intramuscularly.

4:30 p. m.: Semicomatose. Attempts to rise on front legs, but movements are uncoordinated.

10 p. m.: Is able to stand but is rather unsteady.

April 6: Appears entirely normal. Is very active.

April 19: Pulse 100 per minute, irregular.

May 26: 169 days from beginning of experiment and 51 days from the previous attack.

10 a. m.: Seen jumping in kennel; apparently entirely normal.

1:30 p. m.: Found down, unable to rise, conscious, and moderately spastic. The reflexes are hyperactive. Heart rate 70 per minute, the sinus type of cardiac arrhythmia persists. Respirations 18 per minute, deep and regular.

1:35 p. m.: Given 3 mg of riboflavin (L. F. 356) intramuscularly.

1:53 p. m.: Heart rate is 70 per minute; has a marked sinus type of cardiac arrhythmia. Given 1/50 grain of atropin sulfate, subcutaneously.

1:58 p. m.: The heart rate is 228 per minute and regular.

2:40 p. m.: Is very spastic, able to stand on feet when placed on them, but unable to rise.

3 p. m.: Up on feet and walks around; somewhat unsteady. Allowed to wander about, and returned to kennel voluntarily.

May 27: Appears entirely normal. Heart rate is 86 per minute, and there is a sinus type of cardiac arrhythmia.

June 1: Appears normal.

June 28: Heart rate 84 per minute. Animal appears normal.

June 29: 203 days from the beginning of the experiment and 34 days from the previous attack.

10:15 a. m.: Found down, unable to stand or move legs. Heart rate 86 per minute. There is a marked sinus type of cardiac arrhythmia. Respirations 14 per minute and irregular.

11:06 a. m.: Given 2 mg of riboflavin (L. F. 856) intramuscularly.

12:35 p. m.: Had convulsion lasting about one minute, with extreme opisthotonus and involuntary urination.

2:10 p. m.: Is able to walk around, but is rather unsteady.

June 30: Appears entirely normal.

July 30: Appears entirely normal.

July 31: 235 days from beginning of experiment and 32 days from the previous attack.

8:55 a. m.: Found down in cage, unable to rise. Is moderately spastic. Heart rate 80 per minute, quite irregular (sinus type). Respirations 14 per minute.

9:17 a. m.: Heart rate 74 per minute, sinus arrhythmia.

9:37 a. m.: Given 2 mg of riboflavin (L. F. 356) intramuscularly. Had convulsive seizure within 1 minute, with involuntary urination.

12:30 p. m.: Returned to kennel able to stand and run about; is well oriented, but slightly unsteady.

August 1: Very active. Appears entirely normal. Has a sinus arrhythmia. August 19: Appears entirely normal.

August 20: 255 days from the beginning of the experiment and 20 days from the previous attack.

9:00 a. m.: Appeared normal.

2:25 p. m.: Seen standing in kennel in apparently catatoric condition. Moderate general spasticity. Heart rate 64 per minute. Respiration 24 per minute.

3:00 p. m.: Very spastic. Able to stand if placed on feet. Apparently has difficulty keeping eyes open. Heart rate 80 per minute; sinus arrhythmia. Respiration 54 per minute.

3:30 p. m.: Unable to stand; semicomatose. Given 0.5 mg of riboflavin (L. F. 356) intravenously and 1.5 mg intramuscularly.

3:55 p. m.: Spasticity has disappeared. Breathing normally, able to walk but is unsteady.

4:09 p. m.: Heart rate 110 per minute, exaggerated sinus type of cardiac arrhythmia. Appears weak and unsteady, but returns to kennel unassisted. August 21: Active, jumping in kennel. Appears entirely normal.

Discussion.—This dog has been successfully treated with riboflavin (L. F. 356) in five successive attacks of coma. During one of the attacks, % grain of atropin sulfate, administered subcutaneously, immediately stopped the bradycardia and the exaggerated sinus type

of arrhythmis, indicating that these cardiac dysfunctions may be due to a vagotonia.

The first dose of 5 mg of riboflavin (L. F. 356) carried the dog 51 days before a second attack. The second dose of 3 mg of riboflavin (L. F. 356) carried him 34 days before the third attack occurred. The third dose of 2 mg of riboflavin (L. F. 356) carried him 32 days before the fourth attack. The fourth dose of 2 mg carried him 20 days before the fifth attack.

Dog No. 362

December 8, 1936: Begins diet 465 in good condition. Weighs 6.3 kilos.

January 26, 1937: The skin of the chest and the abdomen is quite red. Weighs 8.2 kilos.

January 30: There is redness and partial loss of hair involving the muzzle and the entire ventral surface of the body, including the medial surface of the legs and the axillae.

February 1: Given 72 grams of American cheese by mistake.

February 16: The condition of the skin of the ventral surface of the body is unchanged. Weighs 9.1 kilos.

May 4: Condition of the skin remains unchanged. Weighs 11.8 kilos.

August 17: Weighs 14.6 kilos. 252 days from the beginning of the experiment the animal continues to have a reddened condition of the skin of the muzzle, chest, and abdomen, including the medial sides of all four legs. The hair is thin and short. Otherwise the animal appears entirely normal.

December 11: Found in coma. Given 2 mg riboflavin (L. F. 356).

December 12: Found dead. Typical "yellow liver" at autopsy.

Discussion.—This animal was on the experimental diet a total of 369 days. The dermatitis may or may not have been due to the specific deficiency under consideration. The animal was watched for coprophagy, but this was not observed. It is to be noted that this animal more than doubled his weight while on the experimental diet. The effect of the accidental feeding of 72 grams of American cheese on February 1 is difficult to evaluate. Cheese has been reported by Day and Darby (9) to be a good source of riboflavin.

Dog No. 364

December 8, 1936: Begins diet 465 in good condition. Weighs 4.5 kilos.

April 5, 1937: Has a small ulcerated area on the lateral surface of each foreleg over the wrist joint. Heart rate is very rapid and irregularity makes counting uncertain.

April 12: 125 days from the beginning of the experiment.

7:30 a. m.: Is quite weak, but is able to walk.

8:45 a. m.: Spastic, unable to rise to feet, but when placed on feet is able to stand. The hindquarters seem rather weak.

9 a. m.: Very spastic; unable to walk. "Settles down' gradually when placed in sitting position.

9:15 a. m.: Unable to stand. Moves legs and head with great difficulty. Head can be moved passively with difficulty due to the marked spasticity. Is conscious. Heart rate 118 per minute; regular.

9:20 a. m.: Given 2 mg of riboflavin (L. F. 356) intramuscularly.

10:40 a. m.: The dog is up on his feet, has lost all spasticity, and appears to be entirely normal in every respect. Is very active.

April 13: Appears to be entirely normal.

April 87: Weighs 4.4 kilos. Ulcers over wrist joints have completely healed.

June 14: 188 days from beginning of experiment and 63 days from the previous attack. Appears normal at 9:30 a.m. Heart rate is 158 per minute.

1:45 p. m.: Found down in cage, unable to get up.

2 p. m.: Placed on feet; walked a few steps with extreme difficulty. Heart rate 82 per minute. Respirations 20 per minute.

2:07 p. m.: Given 1 mg of riboflavin (L. F. 356) intramuscularly.

2:35 p. m.: Heart rate is 96 per minute. Neck is rigid; unable to stand; semicomatose.

3:25 p. m.: Given 1 mg of riboflavin (L. F. 356) intraperitoneally.

3:45 p. m.: Able to walk. Heart rate 93 per minute. Respirations 24 per minute.

June 15: Appears to be entirely normal (with the exception of pallor of the buccal mucosa). Heart rate is 147 per minute.

August 17: Weighs 4.3 kilos. In good condition.

Discussion.—This animal has had two attacks of collapse, and each time has made a rather dramatic response to the administration of riboflavin. The first dose of 2 mg carried him a total of 63 days before the occurrence of the second attack.

SUMMARY

All of the five dogs on experimental diet No. 465 have developed a symptom complex similar to that previously described from this laboratory by Sebrell (2, 3) as "yellow liver" and by Sebrell, Onstott, and Hunt (1) as "riboflavin deficiency." The symptoms appeared in dogs No. 347, 357, 360, 362, and 364 in 140, 121, 118, 369, and 125 days, respectively, from the beginning of the experiment. Three of the dogs (Nos. 347, 362, and 357) died in the first attack in spite of the treatment administered. One of the remaining two dogs (No. 360) had five successive attacks of coma, and the other (No. 364) had two attacks of coma. All of these attacks were immediately relieved by treatment with riboflavin (L. F. 356).

The electrocardiogram obtained on dog No. 357 while in an attack of coma was normal with the exception of the extremely slow rate (40 per minute), an inversion of the T-wave, and a regular irregularity of the sinus type. This finding indicates that the cause of the bradycardia and arrhythmia is extracardiac.

One dog (No. 360) was treated with ‰ grain of atropin sulfate subcutaneously during an attack. At the time of administration the pulse rate was 70 per minute and there was a marked sinus type of arrhythmia. Within 5 minutes the pulse rate was 228 per minute and regular. This suggests that the bradycardia and the arrhythmia are vagotonic in origin.

93 January 21, 1988

An analysis of the data given in table 2 indicates that an anemia of the hypochromic type develops in dogs on diet 465. The degree and course of the anemia, as indicated by weekly red blood cell counts and hemoglobin determinations, and the fact that there was no constant and material improvement in the blood picture following the administration of relatively large doses of riboflavin suggests that the anemia may be due to some other factor.

In our experience with riboflavin-deficient diets given dogs in this laboratory, we have never seen collapse occur in less than 102 days. It is necessary, presumably, to deplete the animal's store of riboflavin before the acute symptoms develop. During the depletion period the animals developed an inconstant dermatitis consisting of erythema, followed by a dry, flaky exfoliation. In male dogs, this dermatitis has involved the scrotum; otherwise it is apparently most common on the chest, abdomen, the insides of the thighs, and the axillae.

The symptoms characteristic of the acute attack develop rapidly, and death occurs within a few hours. There is a sudden onset of weakness with ataxia, and very soon the animal is unable to stand or move his legs. At this time there is a varying degree of spasticity, which is at times marked. Apparently the animal is fully conscious, since it will follow activity in the room with its eyes and attempt to move the head or wag the tail. There is no evidence of pain or discomfort. There is bradycardia and an exaggerated sinus type of cardiac arrhythmia (the heart speeding up on inspiration and slowing on expiration), probably due to a vagotonia. The respiration is slow and regular.

The condition advances rapidly, so that within an hour the animal has usually passed into a deep coma, in which there is no response to stimulation. The pulse becomes slower and the breathing more shallow and labored. The animal will remain in this condition for several hours until death intervenes. We have never seen an animal survive more than 12 hours without treatment with riboflavin. Before death the heart rate is slowed even more, being observed as slow as 40 per minute, and the respirations are extremely shallow and labored. Death is apparently precipitated by respiratory failure.

No accurate estimate of the amount of riboflavin necessary for the dog can be made from this data. With the relatively enormous doses given in the treatment of the animals in this experiment, it is probable that some was excreted, and the amount given therefore cannot be considered as being a minimum requirement. Then again, our diet 465 undoubtedly contains a small amount of riboflavin. It is interesting to note, however, that in two dogs receiving doses of 2 mg (the smallest successfully used) one, weighing on an average of 4.4 kilos, was carried for 63 days before the next succeeding attack; and

the other, weighing on an average of 6.9 kilos, was carried for 32 and 20 days before the next succeeding attacks.

CONCLUSIONS

- 1. A symptom complex in dogs, similar to that previously described from this laboratory as "yellow liver," characterized by bradycardia, cardiac arrhythmia, collapse, and coma, rapidly followed by death with characteristic necropsy findings, which include a yellow mottling of the liver and degenerative changes in the central nervous system, has been produced in dogs on a diet low in riboflavin.
- 2. The condition is alleviated by early parenteral administration of riboflavin.

REFERENCES

(1) Sebrell, W. H., Onstott, R. H., and Hunt, D. J.: The treatment of black-tongue with a preparation containing the "filtrate factor," and evidence of ribofiavin deficiency in dogs. Pub. Health Rep., 52: 427 (1937).

(2) Sebrell, W. H.: Fatty degeneration of the liver and kidneys in the dog apparently associated with diet. A preliminary note. Pub. Health Rep., 44:

- 2697 (1929).
 (3) Sebrell, W. H.: "Yellow liver" of dogs (fatty infiltration) associated with deficient diets. Natl. Inst. of Health Bull. No. 162, Part III, page 23. Govt. Printing Office, Washington, D. C., 1933.
- (4) Munsell, Hazel E.: Rice polishings as a source of vitamin B. A preliminary report. J. Home Economies, 21: 124 (1929).
- (5) Cowgill, G. R.: The Vitamin B Requirements of Man. Yale University Press. New Haven, 1934.
- (6) Lillie, R. D., and Sebrell, W. H.: Pathology of yellow liver of dogs. Natl. Inst. of Health Bull. No. 162, Part IV, page 37. Govt. Printing Office. Washington, D. C., 1933.

(7) Zimmerman, H. M., and Burack, E.: Studies on the nervous system in de-

ficiency diseases. II. Lesions produced in the dog by diets lacking the water-soluble, heat-stable vitamin B₂ (G). J. Exp. Med., 53: 21 (1934).

(8) Zimmerman, H. M., Cowgill, G. R., and Fox, J. C., Jr.: Neurologic manifestations in vitamin G (B₂) deficiency. An experimental study in dogs. Arch. Neurol. and Psychiat., 37: 286 (1937).

(9) Day, P. L. and Darby, W. J.: Cataract-preventive vitamin (flavin) in cheese. Food Research, 1: 349 (1936).

TOXICOLOGY OF SELENIUM

V. TOXIC AND VESICANT PROPERTIES OF SELENIUM OXYCHLORIDE 1

By H. C. DUDLEY, Assistant Biochemist, United States Public Health Service

The unique chemical properties of selenium oxychloride have brought this substance into recent public notice although its use as a solvent and plasticizer has been known for some time (1). The pro-

¹ From the Division of Industrial Hygiene, National Institute of Health.

The preceding articles of this series are as follows:

I. A study of the distribution of selenium in acute and chronic cases of selenium poisoning. By H. C. Dudley. Am. J. Hyg., 25: 169 (1936).

II. The urinary excretion of selenium. By H. C. Dudley. Am. J. Hyg., 23: 181 (1936).

III. Determination of selenium in air-gas dust mixtures. By H. C. Dudley. Am. J. Hyg., 24: 227 (1936).

IV. Effects of exposure to hydrogen selenide. By H. C. Dudley and John W. Miller. Pub. Health Rep., 52: 1217 (1937).

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jected use of selenium oxychloride in industry makes advisable a study of its toxic and vesicant action on skin contact.

Selenium oxychloride, SeOCl₂, is a pale yellow liquid, when pure, which gives off fumes of HCl on exposure to humid air. Some of its physical properties are as follows: Molecular weight, 166.11; melting point, 10.8° C.; boiling point at 744 mm Hg, 177.2° C. (decomposition); density, 2.42; vapor pressure, 0.048 mm at 20° C. (calculated).

On contact with water or water vapor, selenium oxychloride rapidly hydrolyzes in the following manner:

Selenium oxychloride is soluble in carbon tetrachloride, chloroform, carbon disulfide, and benzol, forming physical mixtures with these solvents. It is a powerful chlorinating and oxidizing agent, reacting with many inorganic and organic materials, including protein materials, leather, wool, natural and synthetic resins, phenol-formaldehyde resins, and most metallic substances (2, 3, 4).

The studies reported here consist of the determination of the toxic dose for rabbits by skin application, and a determination of the selenium content of the blood and liver of two rabbits receiving amounts of selenium oxychloride sufficient to cause death. In addition, there are presented the results of the application of a small amount of pure SeOCl₂ to the forearm of a man.

A. TESTS ON RABBITS

Accurately measured quantities of selenium oxychloride (Baker's reagent) were placed on a clipped area on the backs of normal rabbits weighing from 1,688 to 3,369 gm. The quantity of the reagent was measured by standard capillary pipettes, 0.1 and 0.01 cc. The reagent was allowed to spread over as large an area as possible. The 0.01 cc portion spread over a circular area approximately 1 cm in diameter; the larger quantities gave areas of application of proportionally larger size. The rabbits, after treatment, were placed in cages and their condition and time of death were noted.

In table 1 are shown the results of the skin applications of SeOCl₂ to clipped areas on the backs of normal rabbits. From these data it is concluded that the amount necessary to cause the death of rabbits is less than 0.01 cc. When computed on a weight basis, the toxic dose of SeOCl₂, by skin application, for rabbits is less than 7 mg/kilo. The difficulties encountered in preventing rapid hydrolysis of the SeOCl₂ when measuring quantities less than 0.01 cc caused the results to be unreliable. For this reason, no figure for the minimum lethal dose of SeOCl₂ is given, except to state that this figure is less than 7 mg/kilo. When SeOCl₂ was dissolved in anhydrous carbon tetrachloride and applied to rabbits in a manner similar to that just

described, moderately severe burns resulted. However, there was no marked or deep tissue destruction as occurred when the undiluted SeOCl₂ was applied, nor was death caused even when proportionate toxic amounts were applied. For these reasons, the results of skin tests made on rabbits, using a SeOCl₂—CCl₄ mixture, are omitted. The results were inconclusive and misleading when compared with the action of undiluted SeOCl₂.

TABLE 1.—Mortality data. Deaths resulting from skin application to rabbits of SeOCl₂

Rabbit No.	Weight	SeOCl ₃ applied	SeOCl ₂ per kilo ¹	Results
D	Grams 1, 945 1, 688 2, 776 2, 788 2, 647 2, 270 2, 330 2, 563 2, 658 3, 369	Ce 0. 20 . 10 . 04 . 03 . 02 . 01 . 01 . 01	Ct 248 143 34. 9 26. 9 18. 2 10. 6 10. 4 9. 4 9. 1 7. 2	Dead in 5 hours. Dead in 5 hours. Dead in 2 hours. Dead in 3 hours. Dead in 5 hours. Dead in 5 hours. Dead in 20 hours.

¹ Calculated from density of SeOCl₂, 2.42.

The course of the severe burns resulting from skin application of SeOCl₂ to rabbits may be summarized as follows:

5 minutes after application: Erythema at site of application.

15 minutes after application: Erythema and swelling around site of application. Red precipitated amorphous selenium at site of application.

- 1 hour after application: Swelling increased. Area of burn depressed, surrounded by dark ring.
- 2-10 hours after application: Same as 1 hour. More severe. Entire back swollen.
- 24 hours after application: No animals receiving 0.01 cc of SeOCl₃ survived as long as 24 hours.

In order to show that the toxic effects resulting from skin burns produced by selenium oxychloride are in part attributable to the selenium absorbed, two rabbits were treated with this reagent and killed 3 hours after application. Samples of whole blood and liver tissues were secured and analyses for selenium made (5, 6). It has been shown that, in acute poisoning due to ingestion of seleniferous materials, selenium is largely distributed in the blood and liver (7).

TABLE 2.—Results of determination of selenium in the blood and liver

Rabbit No.	Weight	SeOCl ₂ applied	SeOCla	Whole- blood selenium	Liver selenium
11	Grams 2, 661 2, 769 1, 797 1, 952	Cc Control Control 0.01 0.03	Mg/kilo 0 0 13. 4 87. 2	Ppm 0 0 .6 2.2	Ppm. 0 0 2.0 4.3

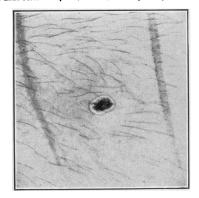


FIGURE 1.—One hour.

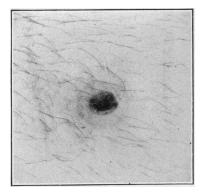


FIGURE 2.—Twenty-four hours.

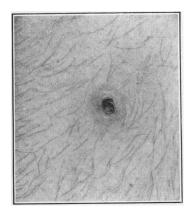


FIGURE 3.—Twelve days.

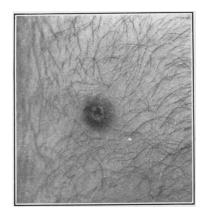


FIGURE 4.—Forty days.

Photographs of burns resulting from the application of SeOCl2 to a man's forearm. (Approximately \times 1.)

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In table 2 are shown the results of the determination of selenium on whole blood and liver tissue of two rabbits receiving 0.03 and 0.01 cc SeOCl₂. These results indicate that the toxic effects of selenium oxychloride are in part due to the selenium introduced into the blood stream through skin absorption.

B. TESTS ON MAN

To the forearm of a man, a minute drop, less than 0.005 cc, of pure SeOCl₂ was applied. The results were noted periodically, and photographs (figs. 1 to 4) were made in order to show the course of the burn and method of healing. In an identical manner, SeOCl₂ was applied to the forearm of the same individual and immediately rinsed with water, in order to show that prompt removal of the reagent with water will prevent serious burns.

The results of skin application of a minute drop of SeOCl₂ to the forearm of a man may be stated as follows:

- 5 minutes: Tissue destruction. Stinging pain. Area of application 1/4 inch diameter.
- 15 minutes: Erythema. Swelling in form of wheal, 1/2 inch diameter. Center of burn depressed.
 - 1 hour: Wheal 1/2 inch in diameter. Radiating erythema 11/2 inch diameter.
- 3 hours: Increased swelling of entire area surrounding burn. Painful. Precipitated Se at site of application.
- 8 hours: Swelling of forearm. Painful. After 8 hours, pain and swelling subsiding.
 - 24 hours: Swelling subsiding. Induration of subcutaneous tissue.
 - 3 days: Swelling subsided. Induration of subcutaneous tissue.
 - 5-10 days: Healing progressed. No secondary infection. Scab formation.
- 10 days: Healing well progressed. No pain. Depressed crythematous area around scab.
 - 10-20 days: Unchanged.
 - 20 days: Slight secondary infection. Treated with tincture of iodine.
 - 25 days: Healing. Infection cleared.
 - 30 days: Healed. Scab off. Scar tissue at site of application.

Photographs (figs. 1 to 4) taken at intervals of 1 hour, 24 hours, 12 days, and 40 days are shown in order to illustrate the course of the wound.

When a small drop of undiluted SeOCl₂ was applied to the skin of the same individual as tested above, and immediately flushed with water, no burn resulted. When using SeOCl₂ it seems advisable to have large quantities of water available for immediate use. A general treatment for burns resulting from SeOCl₂ should include immediate flushing with water, followed by some weak alkali, as sodium bicarbonate or dilute ammonia water.

SUMMARY AND CONCLUSIONS

- 1. Selenium oxychloride, SeOCl₂, is toxic and extremely vesicant. When 0.01 cc is applied to the skin of rabbits, death occurs in less than 24 hours,
- 2. The toxic action of selenium oxychloride is in part attributable to the selenium absorbed, as evidenced by the presence of the element in the blood and liver of animals so treated.
- 3. Selenium oxychloride produces a third degree burn when applied to the skin of man. The burn is painful and slow to heal. Immediate flushing with water will hydrolyze the SeOCl, rapidly so that no burn Treatment with weak alkali, as sodium bicarbonate or dilute ammonia water, is suggested as a general primary treatment.

REFERENCES

- (1) Lenher, V.: Selenium oxychloride as a solvent. U. S. Patent No. 1385081,
- July 19, 1921.

 (2) Lenher, V., Smith, G. B., and Town, G. G.: Vapor pressure of selenium oxychloride. J. Phy. Chem., 26: 156 (1922).

 (3) Lenher, V.: Some properties of selenium oxychloride. Part I. J. Am. Chem. Soc., 43: 29 (1921). Part II. J. Am. Chem. Soc., 44: 1664 (1922).

 (4) Lenher, V.: Preparation of selenium oxychloride. J. Am. Chem. Soc., 42:

- 2498 (1920).
 (5) Robinson, W. O., Dudley, H. C., Williams, K. T., and Byers, H. G.: Determination of selenium and arsenic by distillation. J. Ind. Eng. Chem.,
- Anal. Ed., 6: 274 (1934).

 (6) Dudley, H. C., and Byers, H. G.: Determination of selenium in animal matter.

 J. Ind. Eng. Chem., Anal. Ed., 7: 3 (1935).

 (7) Dudley, H. C.: Toxicology of selenium. I. A study of the distribution of selenium in acute and chronic cases of selenium poisoning. Am. J. Hyg., **23**: 169 (1936).

DEATHS DURING WEEK ENDED JAN. 1, 1938

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Jan. 1, 1938	Corresponding week,
Data from 86 large cities in the United States: Total deaths. Average for 3 prior years. Total deaths, 52 weeks. Deaths under 1 year of age. Average for 3 prior years. Deaths under 1 year of age, 52 weeks. Deaths under 1 year of age, 52 weeks. Deaths under 1 year of age, 52 weeks. Death from industrial insurance companies: Policies in force. Number of death claims. Death claims per 1,000 policies in force, annual rate. Death claims per 1,000 policies, 52 weeks, annual rate.	9, 456 8, 926 446, 524 588 537 28, 485 60, 942, 678 12, 854 9, 6	10, 425 448, 888 685 28, 826 69, 023, 395 11, 799 8, 9

MORTALITY SUMMARY FOR LARGE CITIES, 1937

Provisional number of deaths and infant mortality for a group of 86 large cities of the United States for the 52-week period from Jan. 2, 1937, to Jan. 1, 1938, and comparison with provisional and final figures for 1936

[From the weekly Health Index, Bureau of the Census, Department of Commerce]

	1						ant mo		
	Nu	nber of d	eaths		Numbe	e .		Ra	te
Oity	Pro	visional	Final	Prov	isional	Final	·· Prov	isional .	Final
	1937 1	1986 1	1936	1937 1	1936 1	1936	1937	1936 *	1936
Total (86 cities)	446, 524	448, 888	454, 857	28, 485	28, 826	30, 232	47	50	52
AkronAlbany	2, 245 1, 940	2, 190 1, 932	2, 220 1, 942	202 135	186 130	195 131	50 54	46 58	53
Albany. Atlants. White Negro Other. Baltimore. White Negro Other Brmingham White	4,464	4, 812	4,864	392	419	433	64	74	77
W hite	2, 331 2, 131		2, 529 2, 335	207 185			52 85		
Other	2,101		2,330	100			%		
Baltimore	11, 793	11, 434	11, 516	832	880	893	59	66	67
White	8, 959		8,616	525		.	49		-
Negro	2,822		2,892	307			93		-
Riemingham	12 3, 784	8,771	3,827	369	389	402	74	82	85
	1,905		1,847	200			66		.
Negro	1,877		1,980	169			86		
Other	. 2		. 0	.0			0		-
Boston	11, 552 1, 672	11, 400 1, 676	11,504 1,708	802 105	807 98	822 103	50 41	52 88	52
Bridgeport Buffalo	7,715	7, 594	7,704	524	465	488	- 53	48	50
Cambridge	1, 438	1.422	1.435	114	98	102	44	47	49
	1,720	1 618	1,620	165	151	154	53	49	50
Canton	1, 238	1, 141	1, 162	95	110	110	44	51	53
Cincipneti	36, 217 7, 397	1, 141 37, 900 7, 364	38, 541 7, 452	1, 820 476	1, 851 475	1,885 485	87	39 60	39
Clavaland	10, 297	10, 540	10,620	609	180	610	59 41 53 59	41	52 40 50 49 50 53 39 61 43 62 77
Columbus	4 497	4,672	4,605	250 336	580 285	807	53	58	62
Camden Canton Chicago Cincinnati Cleveland Columbus Dallas White	3,458	3,611	3.660	336	361	887	59	71	77
White Negro	2, 569 889		2,771 887	252 84			56 87		
Other	909		80%	· 07		0	0'		0
Dayton.	2, 933	2, 832	2,861	216	203	208	49	51	55
Denver	4, 725	4, 536	4,673	356	311	345	59	61	63
Des Moines	1,700	1,796	1,996	133	84	147	41	28 52	49
Detroit Duluth	14, 182	14, 712 1, 186	14, 816 1, 199	1, 184 55	1,371 56	1, 420 60	43 29	32	55 63 49 53 34
El Pago	1, 215 1, 496	1, 403	1, 413	275	213	212	105	81	81
Erie	1, 582	1, 510	1,414	109	78	87	45	34	40
Erie Evansville Fall River	1, 344	1,608	1.641	120	108	112	69	63	66
Fall Kiver	1, 591 1, 405	1,603	1,612	84 176	87 202	87 211	41 49	45 56	45 58
Flint Fort Wayne Fort Worth	1, 405 1, 307	1, 406 1, 315	1, 418 1, 325	78	72	211	87	35	38
Fort Worth	1,944	2, 152	2, 142 1, 705	78 163	171	180	55	61	65
White	1, 538		1, 705	126			50		
Negro Other	406		431	37 0			86	- 	
Grand Rapids	1,803	1,718	1. 732	138	116	115	47	45	44
Hartford	2, 162 4, 334	2, 183 4, 253	2, 215 4, 227	115 403	150	159	46	65	42
Houston	4, 334	4, 253	4, 227	403	345	846	58	67	56
White	3, 031 1, 302		2, 905 1, 320	283 120			50 93		
NegroOther	1, 302		4 320	120					
Indianapolis White Negro	5, 637	8, 717	5, 616	407	369	876	65	63	63
White	4, 759		4, 752	334			61		
NegroOther	876		859	73			95		
Jersey City	3, 614	3, 531	3, 560	229	227	236	53	34	35
Kansas City, Kans	1.715	1,888	1,871	98	144	139	47	79	$\widetilde{\mathbf{a}}$
White	1, 344	-, ,,,,,,	1, 483	98 78			43		
Negro	370		387	20			71		
Other Kansas City, Mo	5, 336	5, 628	5, 680	304	310	822	.0 .53	53	56
manusas City, Millions	A 200 (U ₁ U40	a, 100 .	303 ,	310 .	324 1	ω.	ω.	50

See footnotes at end of table.

Provisional number of deaths and infant mortality for a group of 86 large cities of the United States for the 52-week period from Jan. 2, 1937, to Jan. 1, 1938, and comparison with provisional and final figures for 1936—Continued

			:	Infant mortality							
ا ا	Nu	mber of d	eaths		Numbe	•		Rat)		
: City	Pre	visional	Final	Prov	isional	Final	Prov	isional	Final		
। - ह	1987 2	1936 1	1936 *	1937 1	1936 1	1936 2	1937 2	1936 *	1936		
Knowville White Negro Long Beach Los Angeles Louisville White Negro Other Lynn Memphis White Negro Other Miami Mismi	1, 514 1, 196	1, 504	1, 807 1, 233 274	157 129	193	190	70 63	77	86		
Long Beach Los Angeles	318 1,693 17,965	1, 585 16, 650	1.601	28 74 1, 002	81 977 238	86 988 360	137 27 58 30 29 37	33 56 45	35 56 68		
White	4, 318 3, 254 1, 068	4, 093	16, 774 4, 796 3, 720 1, 075	161 139 22	265	800	29 37	10	00		
Lowell Lynn	1,469 1,065	1, 369 1, 052 4, 702	1, 393 1, 063 4, 798	98 37 360	86 30 439	94 38 454	0 49 27 69	72 23 89	5% 3% 9%		
White Negro	4, 421 2, 319 2, 100	4, 702	2, 513 2, 282	202 158		702	64 77				
Mismi White	1, 851 1, 356 490	1, 632	1, 649 1, 198 448	0 124 83 41	101	118	0 51 44 71	48	53		
OtherMilwaukee	5, 570 5, 172	5, 366	5, 409 5, 823	0 291	421 295	428 839	1 0	44	46		
Vashville	2, 679 1, 683 996	5, 713 2, 965	2,978 1,889 1,109	279 235 168 67	254	259	68 64 70	38 67	70		
New Bedford	1, 266 2, 081 8, 005	1,304 2,116 8,719	1, 307 2, 118 8, 811	77 69 749	101 62 825	102 102 834	34 68 64 79 45 33 79 66 97	58 29 92	59 34 90		
White Negro	4, 892 3, 113		5, 237 8, 565	879 370			66 97 0				
Negro Other New York Bronx Borough Brooklyn Berough Manhattan Borough Richmond Borough Richmond Borough Newark, N. J Jakland Nishoma City Jakland Asterson Teogla. Titzburgh Tortland, Oreg Tortland	77, 135 12, 011 26, 004	77, 022 11, 938 25, 900	77, 687 12, 048 26, 067	4, 449 645 1, 674	4, 447 626 1 648	4, 500 625 1, 670	44 41	45 41 42	42 42 42		
Manhattan Borough Queens Borough Richmond Borough	27, 730 8, 975 2, 415	25, 900 27, 946 8, 863 2, 375	28, 303 8, 834	1,597 421 112	1, 648 1, 669 421 83	1,700 421 84	50 38 46	53 40 86 42	53 41		
Newark, N. J Dakland Dklahoma City	5, 038 3, 636 2, 308	3, 027 3, 570	2, 435 5, 106 3, 611 2, 434	267 221 192	83 806 183 167	316 186 238	42 50 38 46 46 50 44 35 66 45 52 36	44	30 44 44		
maha aterson aterson	2, 925 1, 743 1, 568	2, 428 3, 132 1, 739 1, 577	8, 068 1, 764 1 503	172 91 179	222 110 141	226 121 145	44 35 66	52 42 59 47 50	44 64 83 44 61		
hiledelphia ittsburgh ortland. Oreg	25, 186 9, 358 4, 305	1, 577 24, 896 8, 953 4, 192	25, 105 9, 035 4, 224	1, 350 700	1, 387 617 163 206	1, 418 630 173	45 52 36	47 50 87 39	48 51 39 40		
revidence Righmond	4, 305 3, 456 2, 824 1, 688	4, 192 8, 255 8, 216	3, 284 8, 237 1, 882	174 256 222 106	206 231	210 244	47	39 75	40 70		
Negro Other tochester	1, 136 0 8, 731	8, 959	1,352 3 8,997	116 0 166	195	197	105 0 32	39	40		
t. Louis t. Paul alt Lake City	2, 982 1, 894	12, 286 8, 175 1, 764 8, 712	12, 826 3, 307 1, 801	166 463 136 132	419 147 142	641 207 149	84 25 86	83 29 41	80 41 43		
an Antonio White Negro	3, 578 8, 285 290	8,712	3, 728 8, 412 811	604 578 26	620	618	102 102 114	108	108		
Other Rochester	2, 556 9, 244	2, 431 8, 969	2, 431 9, 028	0 112 246	138 282	139 302	82 81	45 89	44 41		
eattleomerville	1, 054 4, 801 895	1, 023 4, 877 962 896	1, 042 4, 919 965	207 46	62 169 44	66 174 46	47 40 88 89 84 89	44 84 87 40 56 53 44	41, 49, 84, 86, 44, 57, 56, 46,		
outh Bendpokanepringfield, Masspringfield, Masspringfieldpringfiel	925 1, 705 1, 875	1, 652 1, 844	919 1, 663 1, 855	65 81 102	61 121 126	67 127 188	84 89	56 53	57 56		
yraciise	2, 675 1, 567	2, 438 1, 585	2, 458 1, 595	157 58	159 71	164 74	29	30	4		

Provisional number of deaths and infant mortality for a group of 86 large cities of the United States for the 52-week period from Jan. 2, 1937, to Jan. 1, 1938, and comparison with provisional and final figures for 1936—Continued

	27	nber of de	41	Infant mortality							
3 14	Nu	HDer of Ge	er (ns		Number	•	Rate				
City	Provisional		Provisional Final		Final	Provisional		Final			
	1937 1	1936 1	1936	1937 1	1936 1	1936 2	1937 8	1936 8	1936		
TampaWhiteNegro	1, 269 918 343	1, 293	1, 316 968 348	94 71 23	79	84	56 52 73	49	51		
Other Toledo Trenton Utica	8 8, 828 1, 885 1, 394	3, 818 1, 890 1, 396	0 8, 840 1, 755 1, 427	261 115 93	215 131 86	223 129 92	0 52 46 50	46 54 45	48 54 50		
Washington, D. C	8, 704 5, 448 8, 241 15	9,031	9, 094 5, 610 3, 469 15	746 342 403	834	847	61 41 102 25	71	73		
Waterbury	941 1, 590 2, 705 1, 241 1, 882	879 1, 485 2, 635 1, 169 1, 889	1, 059 1, 609 2, 653 1, 222 1, 883	74 122 155 71 158	73 108 167 80 127	94 122 168 85 139	37 49 45 43 49	53 51 49 47 42	51 57 51 50 48		

Based upon telegraphic reports received each week from city health officers.
 Calendar year; tabulation of transcripts received from State registrars' offices.
 The provisional infant mortality rate is computed from deaths under 1 year as reported each week, per 1,000 estimated live births for 1936 and 1937, respectively.
 Calendar year; the final infant mortality rate is the number of deaths under 1 year of age per 1,000 live

births.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables a zero (0) is to be interpreted to mean that no cases or deaths occurred

while leaders (.....) indicate that cases or deaths may have occurred, although none was reported.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Jan. 8, 1938, and Jan. 9, 1937

	Dipl	htheria	Infl	uenza	Me	asles		gococcus ingitis
Division and State	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	1 5	7 1 4	13	67	74 42 237 115	99 24 10 930 146 170	0 0 0 2 0 2	0 0 0 8 1
Middle Atlantic States: New York New Jersey Pennsylvania East North Central States:	24 17 87	62 22 50	1 23 22	1 1, 783 118	294 1, 028 2, 633	375 437 83	5 6 1	9 5 5
Ohio Indiana Illinois Michigan ¹ Wisconsin	38 48 19	28 16 80 11 7	7 22 22 22 28	10 346 390 66 655	594 200 2, 627 320 390	31 10 19 19 27	5 1 3 2 1	3 9 2 8
West North Central States: Minnesota Lowa Missouri North Dakota South Dakota Nebraska Kansas	5 4 42 2 12 2 12	12 4 11 2	1 2 118 5 1 10 10	25 2, 854 621 66 42 51 876	7 51 1, 212 81 8 101	38 6 2 5 1	1 4 0 0 0 2 5	2 8 8 0 8 1
South Atlantic States: Delaware Maryland 23 District of Columbia Virginia West Virginia North Carolina 4 South Carolina 4 Georgia 4 Fiorida 4	19 6 19 18 43 2 17	4 18 17 48 12 45 13 15	15 2 66 24 533	8 61 15 76 84 720	6 11 14 199 361 831 168 246	184 230 16 112 28 43 16	0 8 1 5 0 0	• • • • • • • • • • • • • • • • • • •

See footnotes at end of table.

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Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Jan. 8, 1938, and Jan. 9, 1937—Continued

				Infi	uenza	Me	asles	Meningococcus meningitis	
Division and State		Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937
East South Central States: Kentucky Tennessee Alabama 4 Mississippi 3		12	14 13 27 5	69 147 377	318 250	262 361 77	199 9 2	7 6 8 3	19 4 3 0
West South Central States: Arkansas Louisiana Oklahoma 6 Texas 6 Mountain States:		8 13 19 66	2 13 8 86	92 42 87 427	283 47 140 756	49 3 7 51	2 7 14 160	1 5 1 1	2 0 0 2
Montana Idaho Wyoming Colorado New Mexico Arizona Utah ¹		1 12 4 9	7 5 5 5	3 2 106	637 39 77 22 283	8 9 4 174 57 6	102 1 6 10 78	0 0 0 1 1 2 2	4 0 0 1 1 5 0
Pacific States: Washington Oregon California		5 2 45	2 1 30	56 78	7 171 183	17 23 43	126 32 8 126	0 1 2	1 0 10
Total		694	677	2, 423	12, 145	13, 148	3, 956	95	143
	Polion	1yelitis	Scarle	t fever	Sma	llpox	paraty	oid and phoid ers	Whoop- ing cough
Division and State	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	0 0 0	0	26 20 15 240 26 78	22 4 9 228 24 68	0	0 0 0 0	1 0 0 2 0 2	2 0 1 2 0	131 6 50 180 36 49
Middle Atlantic States: New York New Jersey Pennsylvania East North Central States:	1 0 0	2 0 0	549 117 248	687 197 508	0 0 0	3 0 0	4 1 6	5 4 11	349 215 206
Ohio	2 0 1 0 0	0 0 3 2 1	318 190 658 331 181	234 174 473 421 274	6 34 32 0 1	15 12 0 11	1 1 1 2 2	12 2 9 1 0	42 23 104 99 121
West North Central States: Minnesota	1 0 0 0 0 0 1	0 0 0 0 0	132 193 224 26 31 38 201	131 100 126 30 54 37 167	61 55 38 8 5 1	13 33 47 25 2 8 13	0 0 2 0 0 2 2 2	0 1 2 0 1 2 3	66 32 48 25 19 16 104
South Atlantic States: Delaware Maryland ** District of Columbia Virginia West Virginia North Carolina 4 South Carolina 4 Georgia 4 Plorida 4	0 0 0 0 0 2 0 1	0 0 1 0 2 0 0 1	21 54 20 34 75 58 3 18	19 106 18 58 60 52 10 7	0 0 0 0 1 0 0 0 0	0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	0 4 0 2 7 7 4 3	0 2 0 1 2 4 4 3 1	8 55 17 118 72 328 65 9

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Jan. 8, 1938, and Jan. 9, 1937—Continued

	Poliom ye litis		Scarlet fever		Smallpox		Typho parat;	Whoop- ing cough	
Division and State	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Weck ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938	Week ended Jan. 9, 1937	Week ended Jan. 8, 1938
East South Central States:									
Kentucky	0	0	72	63	64	0	1	١.	
Tennessee	ĭ	2	50	50	7	l ŏ	i	8	90 29
A lehame 4	ō	ő	14	22	2	ŏ	3	4	22
Alabama 4	ŏ	ň	13	l ii	ĩ	ľĭ	ň	i	**
West South Central States:	•	•			•	•		•	
Arkansas	1 '	1	13	18	9	0	1	2	22
Louisiana	1	1	10	29	Ō	ì	4	11	
Oklahoma 5	2	0	101	17	9	0	4	2	14
Texas 4	1	1	95	79	0	4	22	9	140
Mountain States: Montana		i	l i						
Montana	0	0	37	35	13	26	1	0	67
Idaho	1	O.	25	28	12	7	3	0	32
Wyoming	0	0	28	18	5	4	0	0	14
Colorado	0	0	33	58	12	3	1	0	10
New Mexico	0	0	16	35	0	0	3	9	19
Arizona	1	0	10	15	0	0	4	1	19
Utah 2	0	0	77	8	0	0	0	0	20
Pacific States:			40			_	_	_	
Washington Oregon	0	1 0	48 41	50 34	23	5	1	3	134
California	2	3	207	285	11 25	27 12	3 13	3 5	10
California			201	280	20	12	13		390
Total	20	21	5, 024	5, 167	459	276	123	137	3, 627

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Menin- gococ- cus menin- gitis	Diph- theria	Influ- enza	Malar- ia	Mea- sles	Pella- gra	Polio- mye- litis	Scar- let fever	Small- pox	Ty- phoid fever
October 1937 Alaska November 1937			24				0		0	0
Alaska Mississippi Wisconsin December 1937	3	97 21	3, 824 199	2, 110	146 298	177	0 13 13	80 851	0 8 14	0 20 4
Connecticut	4 6 10 14	35 64 170 57	14 63 46 127	1 1 24	29 2, 824 1, 904 503	30	1 1 0 2	316 415 241 268	0 0 1 1	3 9 17 6

New York City only.
 Week ended earlier than Saturday.
 Rocky Mountain spotted fever, week ended Jan. 8, 1938, Maryland, 1 case.
 Typhus fever, week ended Jan. 8, 1938, 26 cases, as follows: North Carolina, 4; South Carolina, 3; Georgia, 12; Florida, 1; Alabama, 2; Texas, 4.
 Figures for 1937 are exclusive of Oklahoma City and Tulsa.

Summary of monthly reports from States-Continued

October 1937 Alaska:	Cases	November 1937—Continued	December 1937—Continued
Chicken pox Mumpe Whooping cough	30	Undulant fever: Cases Mississippi	Connecticut
November 1937 Chicken pox: Alaska Mississippi. Wisconsin	428	Whooping cough: Alaska 97 Mississippi 422 Wisconsin 821	Connecticut 2
Dengue: Missis-ippi Dysentery: Mississippi (amoebic)	71	December 1937 Chicken pox: Connecticut	
Mississippi (bacillary) Hookworm disease: Mississippi Impetigo contagiosa:	482	New Jersey 2, 224 North Carolina 1, 013 West Virginia 165 Dysentery:	New Jersey 11 North Carolina 15 Tetanus:
Alaska	39 105 430	Connecticut (amoebic) 1 Connecticut (bacillary) 25 New Jersey (amoebic) 1 New Jersey (bacillary) 1	Trichinosis: New Jersey 1 Tularaemia:
Ophthalmia neonatorum: Mississippi Puerperal septicemia: Mississippi	5 20	Encephalitis, epidemic or lethargic: Connecticut	Typhus fever:
Rabies in animals: Mississippi Rabies in man: Mississippi	18	German measles: Connecticut	Connecticut
Septic sore throat: Wisconsin Trachoma: Mississippi Tularaemua:	2 0 8	Lead poisoning: Connecticut	West Virginia 1 Whooping cough: Connecticut 189
Mississippi Wisconsin	8 1	New Jersey 426	North Carolina 889

PLAGUE INFECTION IN FRESNO COUNTY, CALIF.

Under date of December 28, 1937, Dr. W. M. Dickie, director of public health of California, reported that plague infection had been proved, by animal inoculation and cultural tests, in 29 fleas taken from 5 beecheyi squirrels collected on November 3 in the Shaver Lake area, Fresno County, Calif.

WEEKLY REPORTS FROM CITIES

City reports for week ended Jan. 1, 1938

This table summarises the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and city	Diph- theria	Inf	uensa	Mea-	Pneu- monia	Soar- let	Small- pox	Tuber-	Ty- phoid	Whoop-	Deaths,
Diase and they	C8368	Cases	Deaths	Ca.ses	deaths	fever cases	CBS66	deaths	fever cases	cough cases	C841368
Data for 90 cities: 5-year average Current week 1.	268 162	1, 200 286	156 79	1, 447 2, 539	978 849	1, 572 1, 192	16 43	384 350	26 32	981 742	
Maine: Portland New Hampshire:	1		0	0	8	2	0	1	0	10	36
Concord Manchester Nashua	0		0 1	9 0 0	1 0	0 0 0	0	1 0	0	2 0 0	17 19 4
Vermont: Barre Burlington Rutland	0		0	0 0 0	0	0 0 1	0	0	0	0 7	4 9
Massachusetts: Boston Fall River	1 1		0	51 0	20 5	81 1	0	6	20	6 16	208
Springfield Worcester Rhode Island: Pawtucket	0		1 0 0	1 1 0	7 0	14 4 3	0	2 3 0	0	3 5 0	88 48 65
Providence Connecticut: Bridgeport	Ŏ O	2	0. 1	0	7	15	0	1	0	18 0	17 71 40
Hartford New Haven New York:	0		0	1 0	5 1	17 5	0	0	0	2 4	41 42
Buffalo New York Rochester Syracuse New Jersey:	0 89 0 0	17	1 8 0 0	8 46 4 0	19 125 11 5	18 163 2 2	0 0 0 1	6 72 4 0	0 7 0 0	14 115 4 5	171 1, 465 88 50
Camden	2 1 0	2 5 1	2 0 1	22 4 129	4 8 4	2 12 5	0	2 8 3	0 0 1	1 21 5	45 100 52
Philadelphia Pittsburgh Reading Scranton	3 5 0 1	10	8 3 0	121 301 4 23	36 35 2	83 50 7 3	0	17 6 1	5 1 0 0	23 20 0 0	482 198 19
Ohio: Cincinnati Cleveland Columbus Toledo	1 1 1 0	18 2 1	8 1 2 1	11 99 6 53	15 16 7 6	14 36 6 5	0	5 13 4 4	0	9 27 6 0	157 203 91 80
Indiana: Anderson Fort Wayna Indianapolis Muncie South Bend Terre Hauta	0 2 9 0		0 0 2 1 0	0 0 7 12 0	2 8 16 3 3	3 5 13 3 2	0 0 2 0 0	1 3 2 1 1	0	1 0 1 0 4	17 23 116 14 16 27
Illinois: Alton Chicago Elgin Moline Springfield	0 7 0 0	14	0 2 0 0	5 471 0 29 2	8 55 8 2 0	11 175 7 10 5	0 1 0 0	1 38 0 0	0000	0 23 1 0	10 878 18 6
Michigan: DetroitFlintGrand Rapids	9 2	2	1 0 0	196 0 5	27 6 6	100 25 22	0	16 0 0	8	44 6 7	288 81 35
Wisconsin: Kenosha Madison Milwaukee Racine Superior	0 0 3 0		0	0 2 190 6	0 1 16 1	5 8 20 5 4	0	0 0 1 0 0	0000	2 2 23 4 0	8 10 148 12 13

¹ Figures for Little Rock estimated; report not received.

City reports for week ended Jan. 1, 1938—Continued

	Diph-	Inf	luenza	Mea-	Pneu-	Scar- let	Small-	Tuber-	Ty- phoid	Whoop-	Deaths,
State and city	theria cases	Cases	Deaths	sles cases	monia deaths	fever cases	Pox cases	culosis deaths	fever cases	cough	all causes
Minnesota:											
Duluth	Ō		1	0	3	7	0	2 2	1	8	27
Minneapolis St. Paul	1 0		8	3 2	10	18 5	0 25	1 4	0	0	102 70
Towa:			*	_				-	-	_	٠.
Cedar Rapids	0			0		1	0		0	1	
Davenport Des Moines	0			18 0		1 24	0		0	8	33
Sioux City	ŏ			ŏ		3	lŏ		ŏ	2	
Missouri:								_	_	١.	
Kansas City	1 2	2	1 0	14 0	10	17 1	0	8	1	0	120 21
St. Joseph St. Louis	á		2	468	33	41	6	8	2	2	306
North Dakota:			1 -		i 1						
fargo	0		0	0	0	1 6	0	0	0	0	9
Grand Forks Minot	1		0	0	0	1	0	0	ŏ	4	5
South Dakota:	•		•	-					-	İ	
Aberdeen	0			0		0	0		0	1	
Nebraska:	0		0	0	5	0	0	2	0	0	43
Omaha Kansas:	v		ľ	v	l °l	U	ı v	ا ا	·	"	70
Lawrence	0		0	0	1	1	0	0	0	1	4
Topeka	0	2	1	3 2	2 6	4	0	0	0	12	30 25
Wichita	0	2	0	Z	l °	•	, v	ا ا	U	4	20
Delaware:						_	_		_		١
Wilmington	0		0	0	2	5	0	1	0	5	34
Maryland: Baltimore	1	8	1	2	26	19	0	7	2	38	265
Cumberland	0		Ō	Ō	0	Ö	Ō	į į	Ō	0	17
Frederick	0		0	0	0	0	0	0	0	0	6
District of Colum- bia:											
Washington	5	5	8	8	20	15	0	12	1	8	195
Virginia:			ا ما	_	ا ما	2	o	1	1	8	18
Lynchburg Richmond	1 0		0	0	4 9	6	ŏ	l i	ō	2	82
Roanoke	2		ŏ	i	i	ĭ	ŏ	ō	ĭ	2 2	23
West Virginia: Charleston			1	13	7	1	0	1	0	0	26
Huntington	0	3	1	26	'	ō	ŏ		ö	ŏ	20
Wheeling	ŏ		0	ž	4	2	ŏ	1		4	28
North Carolina:									0	0	
Gastonia Raleigh	0			0	1 4	0	0	0	ŏ	41	34
Wilmington	ŏ		ŏ	0	5	0	0	1	0	9	12
Winston-Salem.	Ŏ		0	0	5	1	0	2	1	10	17
South Carolina:	1	41	1	34	4	2	0	1	0	1	24
Charleston Florence	ō	31	Ô	7	ā	ő	ŏ	ô	ŏ	ō	9
Greenville	Ŏ		ŏ	Ŏ	2	1	Ó	0	0	12	3
Georgia:	1	21	4	81	15	ا و	0	4	0	17	106
Atlanta Brunswick	ō	41	ō	ő	ő	ő	ŏ	ō	ŏ	0	5
Savannah	Ŏ	74	8	0	3	1	0	1	0	0	34
Florida:	1		0	27	1	اه	0	4	0	4	40
Miami Tampa	4	2 3	2	1	3	ĭ	ŏ	i	·ŏ	i	83
A	•	Ĭ		-	_	_					
Kentucky:			o	0	3	0	0	0	0	1	14
Covington Lexington	0	1 2	ĭ	ŏ	3	ŏl	ŏ	ž	ŏl	ô	24
Louisville	3	2	î	52	16	35	ŏ	3	Ŏ	8	96
Tennessee:	ا ۽	ا , ا	ا ا	انف	6	اه	o	0	1	1	31
Knoxville Memphis	0	4	3 0	101	8	8	ŏ	3	δl	10	84
Nashville	3		ĭ	0	9	ŏ	ŏ	ĭ	ŏ	ĭ	64
Alabama:	-		0	8	9	2	0	4	0	٥	79
						72 1	0.4			v 1	19
Birmingham Mobile	1	16	ĭ	ő	i	ō	ŏ	i	Ò	0	38

City reports for week ended Jan. 1, 1938—Continued

State and city	Diph-	Inf	uensa	Mea-	Pneu- monia	Scar-	Small-	Tuber-	Ty-	Whoop- ing	Deaths,
State and city	Cases	Cases	Deaths	Cases	deaths	fever cases	CBASES	deaths		cough cases	Causes
Arkansas:											
Fort Smith Little Rock	0			0		2	0		0	0	
Louisiana:	_	İ	١.	١.		_	١.		i .		
Lake Charles New Orleans	0	17	0	0	0 30	0 5	8	10	0 2	0 7	191
Shreveport	i	17		6	4	2	8	1 1	ő	ó	30
Oklahoma:	•			ľ	· •	-				v	- ~
Muskogee	0			0		2	0		0	0	
Oklahoma City	1		. 1	Ò	5	6	Ó	1	0	0	48
Tulsa	1			0		• 1	8		0	6	
Pexas: Dallas	3	2	2	0	12	3	0	0	0	2	78
Fort Worth	5	-	8	ŏ	16	ŝ	ŏ	ŏ	ŏ	ő	36
Galveston	Ō		0	Ŏ	ž	ĭ	ŏ	ŏ	ŏ	ŏ	15
Houston	8			Ō	13	5	Ŏ	4	Ŏ	Ó	82
San Antonio	1		1	0	11	0	0	8	1	0	78
Montana:					l						
Billings	0		. 0	0	1	2	0	0	0	0	6
Billings Great Falls	Ŏ		ŏ	ŏ	l il	ō	ž	ŏ	ŏ	8	6 7
Helena	0		0	Ō	Ō	Ō	0	Ō	0	8	7
M issoula	0		0	0	0	0	0	0	2	0	10
daho: Boisa			0	0			6	0	0		5
Colorado:			U	U	١٠٠١	١	0	U	יט	0	5
Colorado						- 1					
Springs	0		0	0	5	2	0	0	0	1	. 9
Denver	6		Ō	75	12	20	0	2	Õ	0	95
Pueblo	0		1	0	0	0	0	0	0	0	8
lew Mexico:			_	48		1		_	_		
Albuquerque	0		0	48	2	1	0	5	0	8	10
Salt Lake City.	0		0	1	2	9	0	2	0	4	30
Vashington:		- 1				1					
Seattle	2	l	0	1	9	4	0	8	0	20	111
Spokane	Ō		ŏ	ő	5	3	ŏ	ŏ	ŏl	10	39
Tacoma	0		o l	0	1	8	1	2	0	8	84
regon:	!	- 1									
Portland Salem.	0		0	2	2	7	0	3	8	2	75
alifornia:	٧١			۰		- 1	۰		۱۳	١	
Los Angeles	17	13	3	6	28	29	0	18	ol	17	384
Sacramento	2		0	0	1	70	ŏΙ	ŏ	1	25	28
San Francisco	1	_	1	i i	14	6	Ō	9	Ō	22	182
Dan Francisco	- 1		- 1	- 1	1	٠,	٠,	• !	٠,		200

State and city		gococcus ngitis	Polio- mye- litis	State and city		gococcus ingitis	Polio- mye- litis
	Cases	Deaths	Cases		. Cases	Deaths	Cases
Maine: Portland Massachusetts: Boston. New York: Buffalo. New York. Pennsylvania: Philadelphia. Ohio: Cincinnati. Cleveland	1 0 4 4 2 6 5	0 1 0 1	0 0 0 0 0	Missouri: St. Joseph	0 8 1 0	1 2 0 0	0
Illinois: Chicago Michigan: Detroit. Minnesota: Minneapolis. Lowa: Des Moines.	2 0 0 2	1 0 0	1 1 1 0	Louisiana: New Orleans Shreveport California: Los Angeles Sacramento	0 0	1 1 0 1	1

Encephalitis, epidemic or lethargic.—Cases: Providence, 1; New York, 1; Syracuse, 1; Baltimore, 1; San Antonio, 1.

Pellagra.—Cases: Atlanta, 8; Savannah, 8; Birmingham, 1; Houston, 1.

Typhus fever.—Cases: Savannah, 2; Montgomery, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended December 4, 1937.—During the 2 weeks ended December 4, 1937, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Ed- ward Island	Nova Scotia	New Bruns- wick	Quebec	Onta- rio	Mani- toba	Sas- katch- ewan	Alber- ta	Brit- ish Colum- bia	Total
Cerebrospinal men- ingitis. Chicken pox. Diphtheria. Erysipelas. Influenza. Lethargic encepha- litis. Measles. Mumps. Paratyphoid fever. Pneumonia. Boal et fever. Tub. reulosis. Typhoid fever. Undulant fever. Whooping cough.	2	9 8 15 24 5 6 7 35 11 1	10 	3 304 188 12 7 7 2 302 	2 2 533 28 3 10 1 342 159 1 58 5 7 267 115 15 3 3 103	1 210 2 	1000 33 2 1007 7 155 1100 2 8	1 39 5 3 73 4 3 52	1 164 1 57 194 28 19 3 60 28 5	8 1,370 241 21 89 3 1,176 216 67 91 33 33 315 157 6841

CUBA

Habana—Communicable diseases—4 weeks ended December 18, 1937.—During the 4 weeks ended December 18, 1937, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	1 33 1 3	3	Scarlet fever Tuberculosis Typhoid fever	1 7 1 14	i

¹ Includes imported cases.

1 Per 1.000 live births.

GREAT BRITAIN

England and Wales—Infectious diseases—13 weeks ended October 2, 1937.—During the 13 weeks ended October 2, 1937, certain infectious diseases were reported in England and Wales as follows:

Disease	Cases	Disease	Cases
Diphtheria. Ophthalmia neonatorum. Pneumonia. Puerperal fever.		Puerperal pyrexia	1, 810 21, 167 588

England and Wales—Vital statistics—Third quarter 1937.—During the quarter ended September 30, 1937, 158,647 live births and 100,295 deaths were registered in England and Wales. The following statistics are taken from the Quarterly Return of Births, Deaths, and Marriages, issued by the Registrar General of England and Wales, and are provisional:

Birth and death rates in England and Wales, quarter ended September 30, 1937

Annual rates per 1,000 popula-	Annual rates per 1,000 popula-
tion:	tion—Continued.
Live births 15. 4	Deaths from—Continued.
Stillbirths	Influenza
Deaths, all causes 9. 7	Measles
Deaths under 1 year of age143	Scarlet fever
Deaths from:	Typhoid fever and para-
Diarrhea and enteritis	typhoid fever 0
(under 2 years of	Violence
$age)_{^1}$ 5. 6	Whooping cough
Diphtheria	

YUGOSLAVIA

Communicable diseases—4 weeks ended December 5, 1937.—During the 4 weeks ended December 5, 1937, certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax Cerebrospinal meningitis. Diphtheria and croup Dysentery. Erysipelas Leprosy Lethargic encephalitis. Measles	28 9 1, 265 68 199 1 2 67	6 5 76 5 2	Paratyphoid fever Poliomyelitis. Scarlet fever. Sepsis Tetanus. Typhoid fever. Typhus fever.	24 8 383 7 15 688 6	2 5 11 61

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CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for December 31, 1937, pages 1952-1965. Similar cumulative tables will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China.—During the week ended January 1, 1938, 1 case of cholera was reported in Hong Kong and 3 cases of cholera were reported in Shanghai.

French Indochina.—During the week ended January 1, 1938, cholera was reported in French Indochina as follows: Annam Province, 5 cases; Tonkin Province, 15 cases.

Plague

Brazil.—During the month of November 1937, 5 cases of plague with 3 deaths were reported in Pernambuco, Brazil, making a total of 22 cases of plague with 9 deaths reported in Pernambuco since January 1, 1937, and a total of 34 cases of plague with 15 deaths in all of Brazil for the same period.

Egypt—Asyut Province—Deirout District.—During the week ended January 1, 1938, 1 case of plague was reported in Deirout District, Asyut Province, Egypt.

Hawaii Territory—Island of Hawaii—Hamakua District—Hamakua Mill Sector.—A rat found on December 30, 1937, in Hamakua Mill Sector, Hamakua District, Island of Hawaii, Hawaii Territory, has been proved positive for plague.

Island of Maui—Wailuku District—Puunene.—One death from plague at Puunene, Island of Maui, was reported on January 13, 1938. The death occurred on January 5, in a Filipino laborer, who lived in a sugar company camp 3 miles from the port of Kahului, where sanitary conditions are reported good and the rat population is said to be low. No information was available regarding localities visited prior to illness.

India—Bassein.—During the week ended January 1, 1938, 1 case of plague was reported in Bassein, India.

United States—California.—A report of plague infection in California appears on page 105 of this issue of Public Health Reports.

Smallpox

Siam—Uttradhani Province.—During the week ended January 1, 1938, 35 cases of smallpox were reported in Uttradhani Province, Siam.

Typhus Fever

Netherlands—Rotterdam.—During the week ended December 18, 1937, 1 suspected case of typhus fever was reported in Rotterdam, Netherlands.

Yellow Fever

Colombia—Santander Department—Velez.—During the week ended December 25, 1937, 1 death from yellow fever was reported in Velez, Santander Department, Colombia.

Gold Coast.—During the period December 23-30, 1937, yellow fever was reported in Gold Coast as follows: Agormanya, 1 case; Ho, 2 cases; Keta, 1 case; Yeji, 1 suspected case.

Sudan (French).—During the week ended January 1, 1938, 1 case of yellow fever was reported in French Sudan, locality not specified.

